The New England
Journal of Medicine

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Volume 244 FEBRUARY 1, 1951 Number 5

INTRAPANCREATIC OBSTRUCTION*

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IT CERTAINLY seems fair to say that Opie's[1] explanation in 1901 of the mode of production of acute hemorrhagic pancreatitis has influenced opinion concerning this condition to the present time. He demonstrated at autopsy a common opening into the duodenum of the bile duct and the pancreatic duct in a patient who had died of acute hemorrhagic pancreatitis, and the presence of a biliary calculus in the ampulla of Vater. This converted the bile duct and the pancreatic duct into a common channel, permitting bile to enter the pancreatic duct. This demonstration has associated biliary-tract disease with the production of acute hemorrhagic pancreatitis from that time on, and even today the reflux of bile into the pancreatic duct is considered by many to be necessary in the pathogenesis of the condition. Whereas Jones[2] states that the causes of acute hemorrhagic pancreatitis may be varied, he concludes that the evidence indicates that bile entering the pancreatic duct is the most common cause, and cites the conclusions of Dragstedt, Haymond and Ellis[3] that it is responsible for 60 per cent of all cases seen.

Later, Opie[4] demonstrated the frequency of this anatomic arrangement, and found a common opening of the two ducts in an exceedingly large number of autopsy specimens, 89 per cent, but in only 30 per cent of these was it anatomically possible for an impacted stone to produce a common channel. It is at once obvious not only that the stone must be of proper size but also that the depth of the ampulla must be such that a common channel is actually formed and that both ducts are not occluded. Mann and Giordani[5] reduced this figure to 4.5 per cent of 200 autopsy specimens studied. However, Cameron and Noble,[6] in 66 per cent of their specimens with fresh dissection and the introduction of a small stone into the ampulla, were able to cause regurgitation of bile into the duct of Wirsung. The infrequency with which this occurs, however, is attested by the finding of a gallstone in the ampulla by Schmieden and Sebening[7] in only 4.4 per cent of over 1200 cases of hemorrhagic pancreatitis, and in this country Fallis and Plain,[8] McWhorter[9] and Lewison[10] were able to demonstrate only 9 cases of the condition in 125 patients. It is apparent that the conversion of the common bile duct and the pancreatic duct into a common channel by no means explains all the cases of the disease. Archibald's[11] suggestion was that spasm of the sphincter of Oddi could act in the same manner and produce a common channel. Not only is it necessary for the two ducts to open in a common ampulla but also the sphincter of the common duct must be beyond the junction of the two ducts. Mann and Giordani[5] found the sphincter before the opening of the dual duct in most cases. Furthermore they showed that many of the fibers of the sphincter surrounded the duct of Wirsung as well as the common duct, so that by spasm the sphincter would close both the pancreatic duct and the bile duct. However, Archibald's[11] theory has found its strongest support in the demonstration of Best and Hicken[12] that in the presence of biliary-tract disease Lipiodol injected into the biliary tree through a drainage tube in the common duct will visualize the duct of Wirsung in about 20 per cent of the cases studied. This indicated to them that the normal function of the sphincter of Oddi may be so altered as to permit entrance of bile into the pancreatic duct. However, the mere formation of a continuous channel is not enough: the spasm of the duct sphincter must be sufficiently intense to maintain this abnormal state and the pressure relation must be such that bile will enter the pancreatic duct. Furthermore, it does not offer an explanation for the cases in which the ducts do not open together, with or without biliary-tract disease. More recently, Doubilet and Mulholland[13] have reported relief of recurrent upper abdominal pain associated with increases of serum amylase by cutting the sphincter of Oddi in 28 cases. They indicate that a common passageway is necessary, and imply that such patients are more prone to recurrent pancreatitis, but the frequent focal distribution of pancreatitis found at autopsy does not seem to bear this out.

*The Evans Lecture, delivered at the Robert Dawson Evans Memorial, Massachusetts Memorial Hospitals, Boston, October 18, 1949.

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That the presence of bile in the pancreas will by virtue of its own irritating and necrotizing properties injure the gland is readily admitted by most investigators. The same statement holds true for practically any irritant, as Rich and Duff[14] and others have demonstrated. The flow of bile into the pancreatic duct does require a pressure differential in favor of the biliary side of the common channel that some think exists normally, whereas others believe it is created by the existence of a chronic biliary infection.

With bile in the pancreatic tree, its mode of action must be the rupture of ductules of acini of the pancreas, either by the sheer force of the pressure exerted or by its necrotizing power. It has been shown experimentally that bile injected into the pancreatic duct in sufficient quantities to cause obvious rupture of ductules or acini will produce the disease whereas with small amounts neither necroses nor hemorrhage could be produced by Archibald.[11] The escape of pancreatic juice must take place to account for the fat necroses regularly present in acute hemorrhagic pancreatitis, and bile does not account for the hemorrhage that is a cardinal feature of the disease. Hemorrhage resulting from bile in contact with the vessel wall is never observed in bile peritonitis when there is free access of unlimited amounts of bile to peritoneal vessels. That trypsin can do this has been amply demonstrated by Rich and Duff.[14] Some have suggested that infected bile is potent in this respect, the activation of trypsinogen being brought about by the action of bacteria. But Rich and Duff[14] have demonstrated that sterile trypsinogen injected into the subcutaneous tissues produces vascular necrosis, being converted into active trypsin by substances present in the tissue fluids or liberated from necrotic cells.

As unconvincing as the explanations of the mode of development of acute hemorrhagic pancreatitis were, and as completely as they failed to explain the condition in all cases, it was evident that obstruction must play a prominent role and that only escape of pancreatic juice into the tissues would explain the fat necroses regularly present. It must be remembered that the presence of bile in the pancreatic ducts is evidence of pressure and creates obstruction to the outflow of pancreatic juice, which is passed along through the ramifications of the pancreatic duct system, finally reaching the acini, which must bear the brunt of this obstructing pressure. Rich and Duff,[14] in 1936, demonstrated that an intrapancreatic obstruction was produced by metaplasia of the epithelium of the pancreatic ducts. This metaplasia had been previously observed and described by Priesel[15] in 1922 and Baló and Ballon[16] in 1929 but in neither instance with reference to hemorrhagic pancreatitis. Rich and Duff[14] found evidence of intrapancreatic obstruction as indicated by dilatation of small ducts and acini in 23 of their series of 24 cases and obstructing metaplasia of the duct epithelium in 13 of this series. This metaplasia consisted of a transformation of the normal columnar or cuboidal duct epithelium to a transitional or squamous type of epithelium, which, with proliferation, created masses of epithelium protruding into the lumen and obstructing the ductule in varying degree. The smaller branches of the duct system were involved, and the lesion was distinctly focal in its distribution. This is important in that it indicates that the changes responsible for the damage to the pancreas may be localized and of necessity must have been within the pancreas.

Whereas fat necroses present in hemorrhagic pancreatitis are the result of the action of the lipase of the pancreatic juice, Rich and Duff[14] showed conclusively that the trypsin of the juice was capable of producing necrosis of blood-vessel walls, accounting for the hemorrhagic feature of the disease. They showed that purified trypsin produces rapid necrosis of blood-vessel walls, with resulting hemorrhages, in all respects identical with the vascular necroses regularly found in hemorrhagic pancreatitis. In these lesions no fat necroses were present. With the injection of pancreatic juice into the subcutaneous tissues of animals both fat necroses and vascular necroses were produced. These authors have shown that the trypsinogen contained in pancreatic juice taken directly from the pancreatic duct becomes activated when it comes in contact with tissue fluids quite independent of enterokinase, and is capable of acting in a manner identical with juice activated by the secretion of the intestine. The degree of hemorrhage, therefore, depends upon the size of the vessel rupturing, and it is their belief that a great deal of the death of pancreatic tissue in hemorrhagic pancreatitis represents ischemic necrosis of portions of the pancreas that normally derive their blood supply from the now ruptured vessels. Others have found these characteristic vascular lesions described by Rich and Duff.[14] Weiner and Tennant[17] observed them in cases they reviewed.

Finding metaplasia of the duct epithelium in 23 of 24 of their cases of hemorrhagic pancreatitis and of sufficient degree to produce actual duct obstruction in 13 of the series, Rich and Duff[14] have demonstrated a method of production of acute hemorrhagic pancreatitis that is intrapancreatic, is dependent solely on abnormal pressures created and not only may account for the cases in which no external obstruction is found but does not require that sphincter spasm be invoked to explain an otherwise totally unexplainable situation.

The frequency of occurrence of this metaplastic change is a matter of interest. In addition to their cases of hemorrhagic pancreatitis, Rich and Duff[14] demonstrated it in 28 per cent of 150 unselected autopsies of persons over twenty-five years of age who had died from causes other than pancreatitis.

Priesel[15] and Baló and Ballon[16] found it in 10 and 9 per cent respectively, whereas Yotsuyanagi[18] reported it in 64 per cent of specimens of normal pancreas. Lewison,[10] in his review of the subject, regarded this frequency in the normal pancreas as indicating a lesser importance of this change in the production of hemorrhagic pancreatitis because of the infrequency of the disease. In more than 2500 routine autopsies on persons over twenty-five years of age, I was able to find 81 cases of metaplasia of the pancreatic ducts in varying degrees of intensity. This is an exceedingly small percentage and does not, I am sure, represent its true incidence in this material. This was a routine examination of the pancreas from which one or two sections were made, and in more thorough search for this change, it is not unusual — in fact, it is the rule — to find metaplasia in one or two locations in the gland and not elsewhere.

Its focal distribution is important, for cases of hemorrhagic pancreatitis occur that are distinctly focal in their distribution and it is impossible to explain them except by a focal and limited dissemination of pancreatic juice. One of my cases amply demonstrated this point. It happened that the routine section of the pancreas made at autopsy showed extensive metaplasia of the duct epithelium, dilatation of ductules, and acinar and fat necroses and even hyaline changes in some of the vessels. Pursuit of this finding failed to demonstrate any such changes in more than twenty further sections taken from various locations throughout the pancreas. If the occurrence of duct epithelium metaplasia is as frequent as Yotsuyanagi[18] suggests, it need not be interpreted as militating against its role in hemorrhagic pancreatitis, for the all-important factor is whether or not it creates obstruction. An examination of sections of pancreas showing the change will make it apparent that it does vary markedly in extent and is often extremely localized. That the lesion produces obstruction is attested by the fact that dilated ductules and acini are commonly found distal to the point at which a focus of metaplasia narrows the lumen of a duct. That the rupture of these dilated acini with escape of pancreatic juice may be a sequel is attested by the fact that small focal areas of fat necrosis may be found adjacent to a focus of dilated acini.

Of the 81 cases of duct-epithelium metaplasia reviewed, 49 showed focal dilatation of ductules and acini, and in 14 there were focal areas of scarring unaccounted for by any vascular lesion or other change. In 13 there were focal fat necroses, whereas only 5 gave any evidence of inflammatory reaction, indicating that in most cases the change was not recent. In some, sufficient time had not elapsed for all evidences of inflammation to subside. The sequence of events from evidences of recent damage to old and healed scars can be observed in such a series. In 5 cases there were hyaline and necrotic changes in the walls of pancreatic vessels of the nature described by Rich and Duff,[14] but in none had actual rupture occurred. It must be emphasized that the frequency of ductal and acinar dilatation and fat necrosis in these 81 cases, in which only routine sections of the pancreas were studied, undoubtedly represented a frequency considerably lower than that which could have been found by study of many sections from each pancreas. The requirements for the creation of acute hemorrhagic pancreatitis were fulfilled in these 5 cases, but it would have been difficult to predict its extent.

In this series there were only 10 cases in which either gallstones were present or there were any abnormalities in the biliary tract. In 4 there was inspissated material in the pancreatic ducts or actual pancreatic stones were present. As Martin and Canseco[19] have recently pointed out, such obstruction causes dilatation of ducts and ductules and acini distal to the obstruction, resulting in atrophy and scarring of the pancreas. It becomes evident that obstruction is the all-important factor in the production of ductule and acinar dilatation, and the rapidity with which it forms and whether it is complete or partial determine the course of events distal to it.

In none of these 81 cases showing such changes in the pancreas was death due to the alterations in the pancreas, but, as pointed out above, all the requirements for the development of hemorrhagic pancreatitis existed in 5 cases but the condition had not actually occurred. Twenty-one patients died of carcinoma in various tissues other than the pancreas. The cause of death was hypertensive cardiovascular disease in 10, and rheumatic heart disease in 6. In the remaining 44 cases death was due to 29 different diseases. One can only assume that the factors responsible for this change were inherent in the pancreas and were not extraneous so far as one can tell.

Among the cases reviewed, 51 showed dilatation of pancreatic acini. Of these, 10 revealed dilatation of small ductules, whereas 14 showed metaplasia of duct epithelium in the single section examined. In the same series 63 cases disclosed dilatation of small ductules, and among these 8 demonstrated metaplasia of duct epithelium. It is evident that dilatation of either ductules or acini is not necessarily interchangeable with metaplasia of duct epithelium in this material. The focal nature of duct-epithelium metaplasia has already been pointed out.

It is not easy to transpose these findings to clinical medicine. This is due to inability in many cases to recognize the true nature or source of symptoms. The diagnosis of acute hemorrhagic pancreatitis is often, if it is made at all, an exclusion one, and it is far more frequently made at the autopsy table than clinically.

All physicians are familiar with recurring lower epigastric pain that does not have the character-

istics of gastric disturbances and cannot be shown to be associated with any gastrointestinal lesion or with any biliary-tract disease. It is unassociated with any preceding dyspeptic symptoms, comes on abruptly, often after meals, and may be associated with nausea and vomiting. The course is one of a day or two or three, with complete subsidence, and its nature is never understood. This is not too far removed from the patient with severe epigastric pain, nausea and vomiting, often with evidences of shock and a localization indicating an acute abdominal emergency. On exploration an acute hemorrhagic pancreatitis is found. Efforts

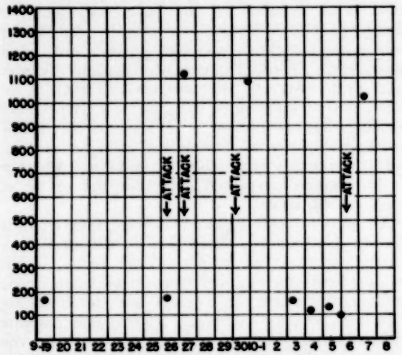


FIGURE 1. *Variations in the Serum Amylase in Relation to Attacks of Abdominal Pain.*

to elicit a history of previous milder episodes are often unsuccessful, whereas a history of more than one attack of hemorrhagic pancreatitis can at times be obtained. The following case is characteristic:

A 37-year-old man entered the hospital for an evaluation of his physical status because of recurring attacks of epigastric pain that had begun 2 years before. These attacks of pain were deep-seated and, he thought, unrelated to meals. At the time of a severe attack 4 months after onset of these symptoms, the abdomen was explored, and the pancreas was found to be enlarged and hard, with surrounding inflammatory reaction but with no stones in the gall bladder. The gall bladder was drained. He continued to have the attacks, with sudden onset of deep-seated pain, always in the same location, and he consumed increasing amounts of alcohol in an effort to relieve the pain when it was present and to prevent its recurrence. A year and a half later, a second operation was performed during an attack; the pancreas presented a similar appearance, and the gall bladder was drained a second time.

The patient was seen after the second operation, when the physical examination disclosed nothing important or pertinent. The blood pressure was 100/60, the blood Wassermann reaction negative, the blood normal, and the urine clear. The gall bladder concentrated the dye satisfactorily, no stones were present, and no stones could be demonstrated in the region of the pancreas. X-ray films of the stomach showed a deformity of the duodenal bulb, but many adhesions had been found at the second operation, and some were released.

Actual hemorrhage was not evident at either operation, and the fact that the patient survived attests that the damage done was not extensive at any time.

This case, unfortunately, does not furnish the proof of the mechanism by which the pancreatitis was produced. The recurrences of the condition indicate that the circumstances responsible for its occurrence continued after the first episode and suggests that they were intrinsic in the pancreas. Whether or not intrinsic obstruction was responsible for the recurring attacks and its exact nature, there is as yet no means of knowing, but the case demonstrates the recurring nature of acute pancreatitis.

With the clinical use of the determination of serum amylase, it was hoped that more light would be thrown upon the many cases of obscure upper abdominal pain. Indeed, such information has been forthcoming, for the serum amylase level is often materially elevated and these significant elevations can only mean that large discharges of the ferment from the pancreas have found their way into the blood stream. Furthermore, the elevation is transient, and its return to normal may mean recovery or may indicate that complete necrosis of the gland has taken place. Also, its level in the blood has no prognostic value, and one has only to examine surgical records of operations in which the gland has been traumatized in any way to appreciate the levels attained by even relatively minor procedures. In the natural course of disease it has been found that the serum amylase remains within constant limits in practically all types of disease except in conditions in which the pancreas is damaged. It does, then, locate the disturbance in an organ that has hitherto received consideration usually by exclusion. It is reasonable that varying degrees of severity may occur in acute pancreatitis, and the determination of serum amylase enables one to recognize many more of these more mild episodes, as the following case demonstrates:

A 43-year-old woman complained of recurring upper abdominal pain of 5 years' duration. There were episodes of severe epigastric pain with nausea and vomiting, coming on shortly after eating or during the night, which at first occurred at intervals of 2 or 3 months. The condition was diagnosed as gall-bladder disease, and after 6 months, the gall bladder was removed.

The gall bladder was said to have been distended and covered with recent adhesions and contained faceted stones. The bile duct was normal, no stone was present, and there was no evidence of peptic ulcer.

The operation afforded no relief; attacks of the same character continued but became increasingly more frequent until they were occurring every few days.

When she entered the hospital, the temperature was 99.6°F., and the blood pressure 100/60. There was evidence of weight loss; the lungs were clear, and the heart was normal. There was tenderness in both upper quadrants of the abdomen, but no masses were felt. Pelvic examination showed only a small cystocele and rectocele.

The serologic test for syphilis was negative; there was no anemia, and the white-cell count was 6000. The stools contained no blood or parasites and were never fatty. The urine

was normal, and the serum bilirubin was less than 0.8 mg. per 100 cc. X-ray films of the gastrointestinal tract were normal; numerous spot films failed to show any pancreatic stones, and a gastroscopic examination was negative.

In the ward, the patient had four attacks of abdominal pain, none of more than a few hours' duration. The first attack occurred ½ hour after the evening meal, and the second ¾ hour after a feeding, and the time relation of the remaining two attacks was not recorded. Numerous serum amylase determinations were made, and their relation to the attacks is presented in Figure 1.

So far as could be demonstrated clinically, this woman's difficulty lay in the pancreas, and although abnormalities existed in the gall bladder, its removal had no effect upon the attacks of pain. They recurred even more frequently and, as the determinations showed, each time with a significant rise in serum amylase. The opportunity to confirm this by exploration was lost since the patient refused operation, but I think the surgical procedure previously carried out removes the biliary tract as the seat of the difficulty. It does show, perhaps conclusively, that attacks of pancreatitis recur in varying degrees of severity and may recur over and over again without a fatal termination.

It can readily be seen how difficult it is to present incontrovertible evidence to substantiate such a chain of events both because of the minute size of the initial lesion and because the damage resulting from the rupture of arteries rendered necrotic by escaped trypsin is often so extensive that the tissue is destroyed by the time one has the opportunity to study it. It therefore becomes necessary to eliminate other possible causes and to arrive at the conclusion in an indirect manner, as undesirable as this reasoning may be. The following case is typical:

The patient (G.M.–U77263), was a 37-year-old man who was known to be an alcohol addict. He was admitted to the hospital in a semicomatose state. No adequate history could be obtained, but it was known that his consumption of alcohol for a period prior to admission had been large.

On physical examination there was little to be found but consolidation of the right lower lobe of the lung and resistance and tenderness in the upper abdomen. The temperature was 103°F. Later, he had a convulsion, and these recurred from time to time until his death 2 days later.

At autopsy (15178), in addition to pneumonia, nodular cirrhosis of the liver was found. The gall bladder and common duct were normal, and there were no gallstones. Over the surface of the pancreas there were many areas of fat necrosis (Fig. 2*B*). On microscopical examination there were many blood vessels within the pancreas with necrotic walls but without rupture or obvious hemorrhage (Fig. 2*D*). In the available material from a routine autopsy little metaplasia of the duct epithelium (Fig. 2*A*) was found, but the change was definite and many of the ductules and acini were dilated (Fig. 2*C*).

In this case the existence of duct metaplasia with dilatation and rupture of acini is evident, and with the exception of moderate cirrhotic change of the portal type no abnormality in the liver or biliary tract was found. Whereas in the routine examination only small amounts of metaplasia of the duct epithelium were found, no thorough search for such a change was made. However, smaller ducts were obstructed, as indicated by the focal ductal and acinar dilatation. Pancreatic juice from the duct-acinar system had escaped, fat necrosis resulting from escaping lipase had occurred, blood vessels had been attacked in the manner characteristic of the action of trypsin, and although actual rupture of such vessels had not yet taken place, they were prepared for rupture, to add the hemorrhagic feature to complete the picture. The only mechanism found by which this state of affairs could be produced were the evidences of intrapancreatic obstruction demonstrated.

In the following cases the metaplastic abnormality was associated with a variety of conditions.

Case 1. L. F. (U–101349) was a 62-year-old patient with mild diabetes whose complaint was right-flank pain and who died of a general peritonitis resulting from the rupture of a perinephritic abscess. At autopsy (15217) the section of pancreas showed many dilated small ducts and acini and a well defined epithelial metaplasia (Fig. 3*A*).

Case 2. E. M. (U–196650), a 50-year-old man, died of a streptococcal septicemia with multiple metastatic lesions. At autopsy (16852) the pancreas showed metaplasia of the pancreatic-duct epithelium with dilatation of the ductules and with fat necrosis of the pancreas (Fig. 3*C*).

Case 3. T. H. (U–227782), a 49-year-old patient, was admitted to the hospital in extreme cardiac failure with arteriosclerosis and hypertension and died in less than 24 hours. At autopsy (17416) the pancreas showed, in addition to early squamous metaplasia of the duct epithelium, ductules dilated and filled with eosin-staining material, and there was focal acinar dilatation. Both conditions could have contributed to or have been responsible for internal pancreatic obstruction (Fig. 3*B*).

Here alteration in the physical character of the secretion enters as a possible cause of duct obstruction. How these alterations are brought about, whether they are cause or effect in duct obstruction, and whether they may even lead to calculus formation are equally obscure. If calculi result from such a change, it is suggested that the attacks of pain associated with pancreatic stone may at times find their origin in rupture of acini and escape of pancreatic juice rather than in simple movement of the stone within the duct.

In the following cases varying degrees of obstruction accompanied the metaplasia:

Case 4. H. T. (U–245299), a 62-year-old patient, died of a carcinoma of the bladder. At autopsy (17705) the pancreas showed definite squamous metaplasia. There was, however, little evidence of obstruction in the routine section, though a few scattered acini were dilated (Fig. 3*D*).

Case 5. S. W. (U–378231), a 53-year-old woman, died of subacute necrosis of the liver. She had had an attack of epigastric pain four months before death and a second attack 3 weeks later. It was 2 months later that she became jaundiced. At autopsy (19897) the liver was small, and there were bilirubin stones in the gall bladder. Careful dissection of the biliary tract showed no stenosis or occlusion of any sort. The pancreas was firm but on section appeared grossly normal, and the pancreatic duct was patent. Microscopical examination demonstrated widespread minute scarring, and some ducts showed metaplasia and dilatation of acini; in one area there was some fat necrosis (Fig. 4*A*).

Case 6. A. M. (U–371647), a 37-year-old woman, an alcohol addict, died of extensive second-degree and third-degree burns. At autopsy (19748) the pancreas showed focal atrophy and scarring, occlusion of two ducts by concretions, and squamous metaplasia of duct epithelium. There

were no other alterations in the gland in the section studied (Fig. 4*B*).

of squamous metaplasia of the pancreatic ducts, with dilatation of acini and focal atrophy and scarring (Fig. 4*C*).

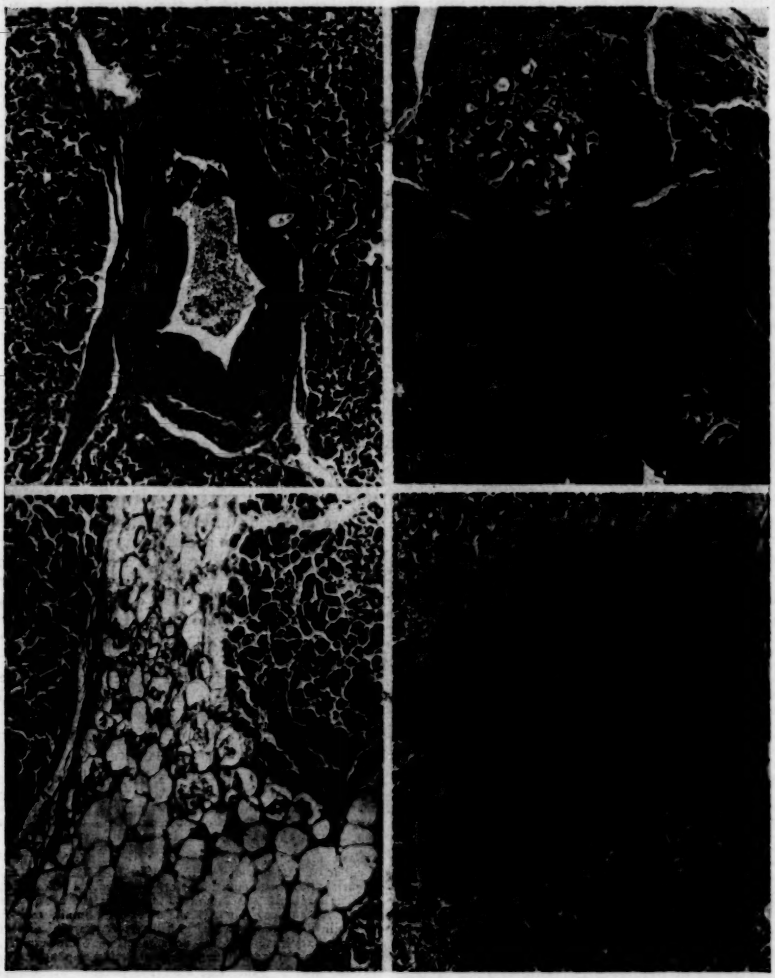


FIGURE 2. *Sections of the Pancreas in a Case of Intrapancreatic Obstruction.*
A = *squamous metaplasia beneath the normal duct epithelium in a dilated duct.* B = *area of fat necrosis within the pancreas.* C = *localized area of dilated acini.* D = *a blood vessel, with partially necrotic wall, which has not yet ruptured.*

CASE 7. H. S. (U–328938), a 54-year-old patient, died of carcinoma of the lung. At autopsy (19137) there were foci

CASE 8. A. S. (U–273583), a 50-year-old patient, suffered from postrheumatic valvular heart disease and died in con-

gestive failure. At autopsy (18248) the pancreas demonstrated marked squamous metaplasia of the duct epithelium; at least one of the ducts was completely obstructed. There were areas of acinar dilatation (Fig. 4*D*).

Case 9. E. T. (U–135004), a 49-year-old patient, died of septicemia due to a Type III pneumococcus after pneumonia and purulent pleurisy. At autopsy (15737) the pancreas showed squamous metaplasia of the duct epithelium, with

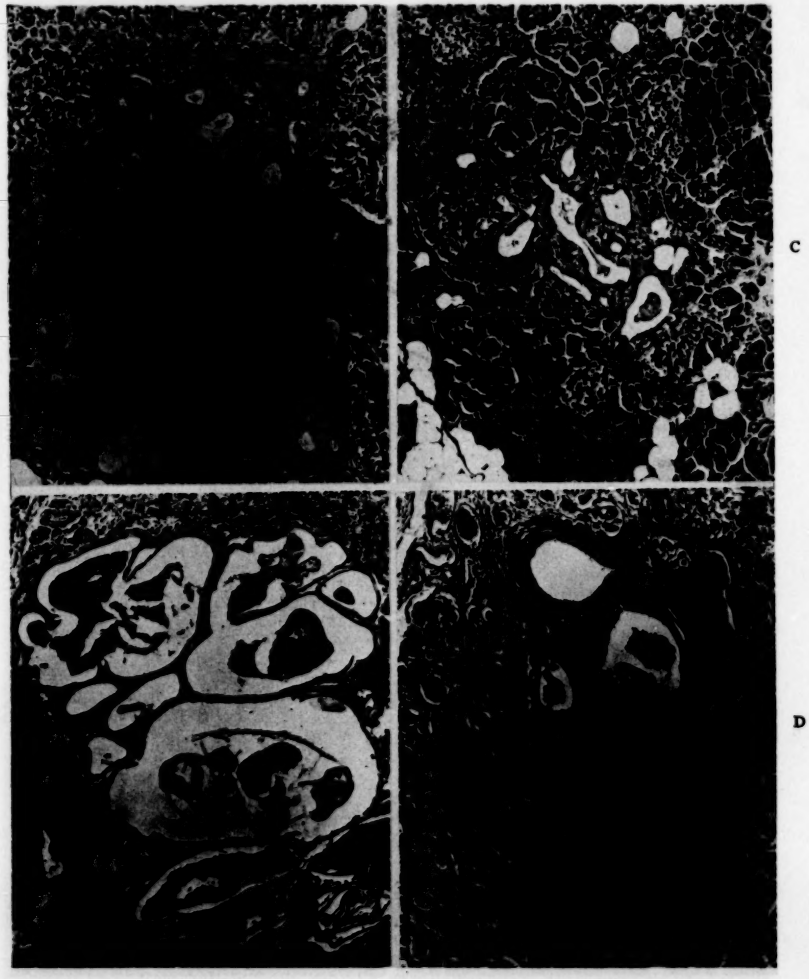


Figure 3. *Sections of the Pancreas in Cases 1–4.*

A = *dilated small pancreatic ducts, some acinar dilatation and squamous metaplasia of duct epithelium in Case 1.* B = *localized area of marked dilatation of pancreatic ducts, filled with inspissated material, and early squamous metaplasia of pancreatic-duct epithelium in Case 3.* C = *well defined metaplasia of pancreatic-duct epithelium with dilatation of ductules in Case 2.* D = *definite squamous metaplasia of pancreatic-duct epithelium, with only slight dilatation of ductules, in Case 4.*

acinar dilatation and areas of fat necrosis and scarring (Fig. 5*A*).

under a variety of conditions. Although all stages of its development are seen, varying degrees of

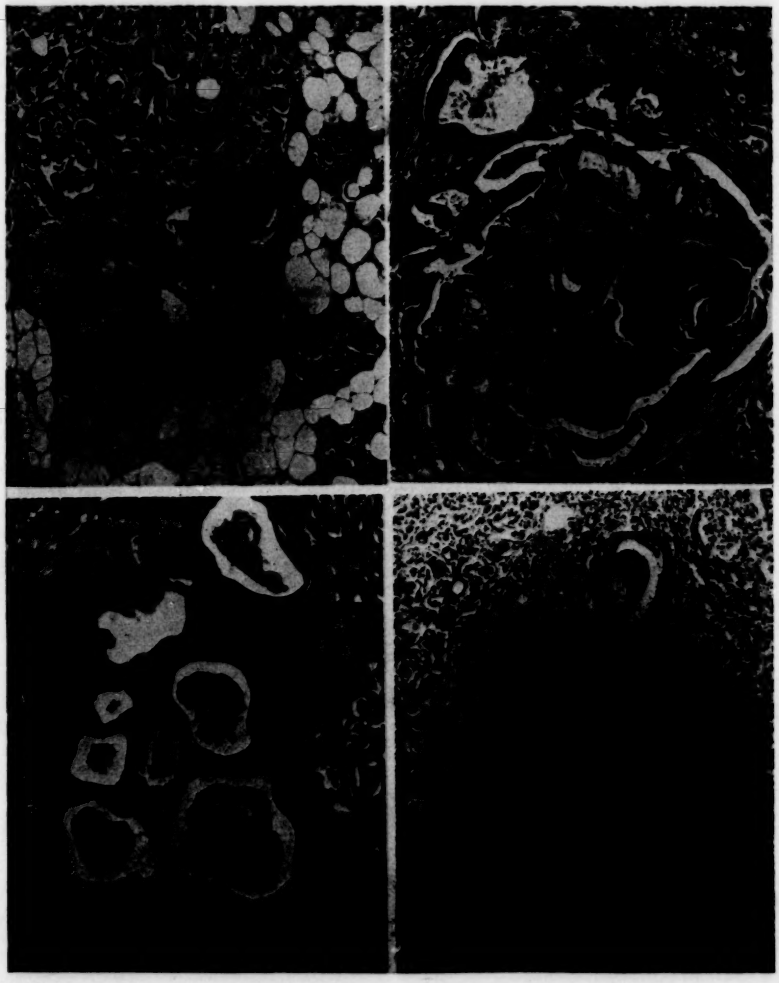


FIGURE 4. *Sections of the Pancreas in Cases 5–8.*

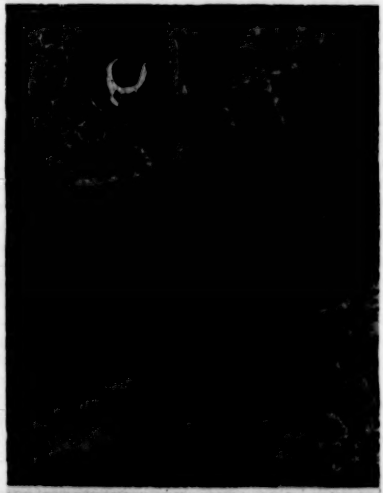
A = squamous metaplasia of pancreatic-duct epithelium and acinar dilatation in Case 5. B = dilatation of pancreatic ducts with squamous metaplasia of duct epithelium of some ducts, and surrounding atrophy and scarring in Case 7. C = squamous metaplasia of pancreatic-duct epithelium and occlusion of duct by concretions in Case 6. D = marked squamous metaplasia of pancreatic-duct epithelium with complete obstruction of one duct by squamous epithelium, and some acinar dilatation in Case 8.

As these cases are reviewed it is evident that this metaplastic condition is found in the duct epithelium

obstruction accompany it. It is also evident that any circumstance occluding ducts produces the

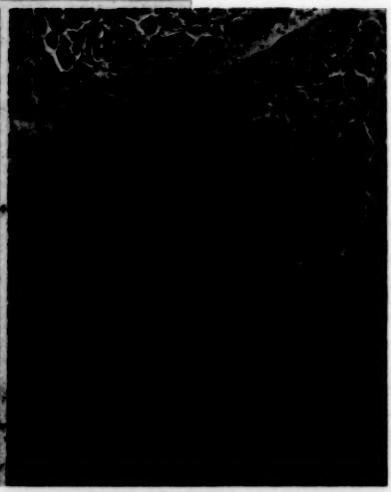
same intrapancreatic obstruction, in which the potentialities of the condition lie.

Case 10. M. T. (U-49571), a 39-year-old woman, who was a chronic alcohol addict, was admitted the day after



A

B



C

Figure 5. *Section of the Pancreas in Cases 9 and 10.*
A = marked squamous metaplasia of pancreatic-duct epithelium, with some scarring and an adjoining area of fat necrosis, in Case 9. B = squamous metaplasia of pancreatic-duct epithelium, cellular infiltration and portion of an adjoining area of necrosis in Case 10. C = necrosis of the wall of a small artery, partially digested but unruptured, in Case 10.

In the following case the obstruction was obvious at autopsy.

delivery and died on the following day in collapse. No diagnoses had been made.

At autopsy (13170) the pancreas was swollen, and throughout the gross section revealed small hemorrhages and areas suggesting fat necroses. The gall bladder and biliary tract were normal, and no stones were present. Microscopical sections of the pancreas showed numerous fresh fat necroses with considerable inflammatory reaction. In several ducts metaplasia of the epithelium was present, with dilatation of acini (Fig. 5*B*), and necrosis of the wall of a small artery partially digested but unruptured was seen (Fig. 5*C*).

In this case the cause of death was acute pancreatic necrosis with hemorrhage. There was no history of previous attacks of abdominal pain, although the patient was a chronic alcohol addict. The gland showed definite metaplasia of the duct epithelium, and distal to this were the evidences of the obstruction created, the ductule and acinar dilatation. No other change that could explain the acute alterations in the pancreas was found, and it seems obvious that obstruction to outflow of pancreatic juice was the fundamental mechanism of this acute pancreatitis, and furthermore that the cause of the obstruction was intrapancreatic.

Discussion

Unfortunately, these matters do not lend themselves to statistical analysis, and the inadequacy of the history may be responsible for this in large part. It is not difficult to understand that when the condition at hand deals with some totally unrelated problem, minor abdominal pain is overlooked by the patient, if the history is not inquired into casually by the examiner. This is disconcerting, for it prevents the physician from closely linking the clinical observation and the post-mortem evidence and compels him to rely upon circumstantial evidence, but evidence that is considered too implicating to be ignored. Although rupture of a pancreatic acinus, with the spilling of minimal amounts of pancreatic juice, may give no symptoms that the patient is aware of, focal lesions of rather larger size produced in this manner probably cause symptoms, misinterpreted though they might be.

The natural sequel of obstruction is increase in pressure in ducts and acini distal to the obstruction. The dilatation of both ducts and acini provides evidence of obstruction and, when focal in distribution, can mean only that the obstruction is within the gland and, indeed, limited to a portion of the gland. The amount of pancreatic juice that escapes into the tissues in and about the pancreas, the concentration of the ferments present and the chance contact of escaping juice with sizable blood vessels determine the nature and the extent of the damage done. Rich and Duff[14] have demonstrated that trypsinogen that escapes into the tissues becomes activated and exerts a specific necrotizing effect upon blood vessels, the size of the vessel so attacked determining the extent of hemorrhage and the extent to which pancreatic tissue will undergo necrosis as a result of deprivation of blood supply.

The fact that obstruction is the primary factor in producing pancreatic necrosis or hemorrhagic pancreatitis seems inescapable. Although obstruction may be produced in many ways, the existence of intrapancreatic lesions capable of producing this obstruction is the phenomenon that demands attention. It is not only another cause of pancreatic-duct obstruction but also the only way in which many hitherto unexplained cases of hemorrhagic pancreatitis can be accounted for. The mechanism first described in the production of acute pancreatitis has so fastened itself upon medical thinking that it has blinded us to the possibility that some mechanism other than biliary-tract disease can produce the same condition. That acini rupture far oftener than is realized clinically is evident from the focal fat necroses found at autopsy adjacent to areas of focal acinar dilatation. It is probable that such ruptures often cause no symptoms; at other times they may cause pain that is attributed to something else, or simply not identified. The recognition of these focal acinar ruptures is clinically difficult, and the measurement of serum amylase is the only precise aid available. Already this test has called attention to the probability that obscure upper abdominal pain can be associated with pancreatic disturbance more often than one realizes, and doubtless the recognition of these pancreatic disturbances will become more frequent as time goes on.

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CEREBRAL PSEUDOTUMOR OR INTRACRANIAL HYPERTENSION OF UNKNOWN CAUSE*

Report of Three Cases

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INCREASED intracranial pressure as measured by spinal puncture or ventricular tap may be the result of any one of several conditions. The more common causes are excessive secretion of the choroid, obstruction secondary to congenital defects, neoplasm or inflammation and deficient function of the spinal-fluid-absorptive mechanism. Masses within the cranium, such as abscess, neoplasm and the hematomas, subdural, extradural and intracortical, also result in intracranial increased tension. Tension may be raised by brain edema secondary to trauma, thrombosis of dural sinuses, hypertensive states and brain-stem compression due to herniation of the temporal cortex through the incisura of the tentorium. Finally, extracranial mechanisms such as right ventricular failure and mediastinal tumor may produce increased intracranial tension.[1]

The cause of increased intracranial pressure is readily determined in most cases. However, attention has been called to a syndrome that has been named cerebral pseudotumor or intracranial hypertension of unknown etiology.[1-4] Its features consist of signs and symptoms of an intracranial space-occupying lesion that cannot be demonstrated by diagnostic procedures such as ventriculography, encephalography and arteriography.

It is our purpose to present the clinical facts and laboratory findings in 3 cases studied at Cleveland City Hospital and suspected of representing the syndrome of cerebral pseudotumor.

CASE REPORTS

CASE 1. F. B., a 36-year-old Negro, was admitted to the hospital on April 23, 1946, complaining of headache, vomiting and dizziness of 7 days' duration. The patient had been well until the onset of his present illness.

Physical examination revealed moderate lethargy, bilateral papilledema, slight nuchal rigidity and absent abdominal reflexes. The remainder of the neurologic examination was negative. There was no fever, and the usual laboratory work was normal. The spinal-fluid pressure was equivalent to 760 mm. of water; the fluid was crystal clear and contained 7 cells per cubic millimeter. Routine films of the skull were normal. After 36 hours' observation the patient became more lethargic, and ventriculograms were made. Radiograms revealed an apparent slight defect in the frontal horn of the right lateral ventricle, and for this reason a transfronto-temporal craniotomy was carried out. The brain was found to be under increased pressure, but exploration of the right frontal lobe, right lateral ventricle, foramen of Munro and third ventricle failed to reveal tumor. The wound was closed, and the patient returned to the ward. Postoperatively his condition deteriorated steadily, and he died on the 3rd postoperative day. Post-mortem examination of the brain failed to reveal any significant abnormalities. The final anatomic diagnoses were bronchopneumonia and recent exploratory craniotomy. The final note of the protocol stated that the cause of the disorder was not determined at autopsy.

Craniotomy was carried out in this case because the clinical history and findings were highly suggestive of brain tumor. When the ventriculograms were found to be slightly confirmatory, with a very small defect in the frontal horn, the procedure seemed well justified. However, no tumor could be found, and the patient died on the third postoperative day.

CASE 2. S. H., a 21-year-old woman, was admitted to the hospital on April 17, 1947, complaining of headache, dizziness and occasional vomiting of 1 month's duration. She was referred from the eye clinic after seeking relief for blurring of vision. The past history was noncontributory.

Physical examination was not remarkable except for bilateral papilledema. There was no fever; laboratory findings were normal. The spinal-fluid pressure was above 600 mm. of water; the fluid was crystal clear with 8 cells per cubic millimeter and 30 mg. of protein per 100 cc. Ventriculograms on the 5th hospital day were normal. Because of recent disastrous experience with Case 1, it was decided to pursue a conservative course. However, the development of slight facial paresis and increased lethargy 24 hours after ventriculography made subtemporal decompression advisable. A right subtemporal decompression was performed, and the dura mater opened widely. The brain bulged markedly, and pulsations were decreased in amplitude. Postoperatively the patient made an uneventful recovery. She became asymptomatic, and the papilledema resolved. Pneumoencephalograms on the 34th hospital day were normal, and she was discharged on the 39th hospital day. Follow-up examination after 18 months showed her to have remained asymptomatic during this period.

This patient was admitted to the hospital with the diagnosis of brain tumor, but more considered study enabled us to avoid extensive exploratory craniotomy. Simple subtemporal decompression controlled the symptoms and restored vision.

CASE 3. J. B., a 23-year-old man, was admitted to the hospital on January 20, 1948, because of throbbing intermittent occipital headaches since the age of 14. In 1942 he had been treated at another hospital for this complaint; repeated lumbar punctures showed only increased spinal-fluid pressure. X-ray films of the skull and air encephalograms in 1942 and 1947 revealed no abnormalities. During the interval since 1942 the patient had suffered with repeated attacks of severe headaches, frequently associated with nausea and vomiting. He was able to pass the physical examination for service with the armed forces and served 9 months in 1942 until released because of recurrent headaches. He was unable to associate the attacks with any particular stimuli except that they were likely to occur during fatigue or emotional stress.

Physical examination revealed bilateral choked disks and some lateral and rotatory nystagmus. Lumbar punctures disclosed maximum pressures equivalent to 760 mm. of water, with crystal-clear fluid and 20 mg. of protein per 100 cc. Ventriculograms on February 17 were normal, but a right

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subtemporal decompression was done to relieve the increased pressure.

The patient continued to have symptoms. Bilateral cerebral arteriograms on April 19 and May 24 failed to reveal any abnormalities in the anterior and middle cerebral circulations. Electroencephalograms on June 2 were normal.

Because the patient continued to have attacks of severe headaches and elevated pressures during the attacks, with bulging of the decompression defect, a left subtemporal decompression was done on June 3. He was discharged on June 10. On August 13 he was readmitted because of persistence of symptoms, and the right subtemporal decompression was enlarged. He was again hospitalized between October 21 and December 2, but symptomatic care and stellate-ganglion blocks failed to induce any relief.

On December 17 he was admitted to another hospital with the same complaints. He was treated with Ergotrate, histamine, Dilantin and Mesantoin, without relief. Because each episode of headache continued to be accompanied by increased spinal-fluid pressure and bulging of both decompression defects, an additional decompression in the suboccipital region was carried out. During this procedure the fourth ventricle and aqueduct of Sylvius were uncovered and explored. No abnormalities were noted. The patient was discharged on July 8, 1949, with slight improvement.

Despite three decompressions this patient's intracranial pressure is still capable of rising sufficiently to produce severe headache. The prolonged course and continued normal diagnostic procedures make it very unlikely that a tumor is present.

Discussion

In these 3 cases no cause for the persistent or intermittent increased intracranial pressure could be determined. Certain characteristic features were noted in each. The signs and symptoms were similar and referable to increased intracranial pressure: lethargy, severe headache, blurring of vision, vomiting and papilledema. The spinal-fluid pressures were highly elevated, and the fluid was crystal clear, without great increase in cell count. There was no fever, leukocytosis or other evidence of inflammation. The ventriculograms revealed no significant abnormalities. The most likely diagnosis in each case seemed to be brain tumor, but there were no localizing signs. The failure of post-mortem studies in 1 case and the course of the other patients lead us to believe that these patients represented the disease syndrome spoken of as cerebral pseudotumor or intracranial hypertension of unknown cause.

Dandy[2] reported 22 cases of this syndrome in 1937, and Sahs[3] added 5 cases in 1939. Davidoff[4] described several similar cases under the title of serous meningitis in 1936. Recently, Yaksin and his associates[5] reported 12 more cases.

Dandy[2] defined the syndrome as the signs and symptoms of a space-occupying lesion that could not be demonstrated. In each of the series of cases reported the features of the syndrome are essentially the same: papilledema, normal ventriculograms, increased spinal-fluid pressure and normal fluid.

The etiology of the syndrome has not been clearly established. Dandy[2] observed his patients for many years and found that the pressure remained elevated a few months to several years and, further, that the pressure underwent rapid changes in any patient depending on certain factors such as emotional reactions. He reasoned that increased pressure due to increased cerebrospinal fluid was not the cause because the ventricular system was normal in size and because evacuation of fluid from the subarachnoid space did not improve the condition. He concluded that variations in the blood volume of the brain secondary to vasomotor changes were responsible for the increased pressure.

Sahs[3] postulated that edema of the brain was the best explanation — edema that evolved rapidly but subsided slowly. He discounted any inflammatory changes because the cell count was usually normal.

Of interest regarding possible etiologic factors in these cases is the report by McCullagh.[6] This case was considered to represent pseudotumor because of normal air studies in the presence of increased spinal-fluid pressure, headache and papilledema. Subtemporal decompression appeared imperative, but further study seemed to prove fairly conclusively that the entire phenomenon was on the basis of menstrual edema. Low-sodium intake and extract of pregnant urine controlled the process fairly well in this patient without surgery.

It is our opinion that the diagnosis of pseudotumor of the brain should be reserved for cases that have been carefully and repeatedly studied to exclude the more common causes of increased intracranial pressure.

This diagnosis must not be used as a justification for inadequate or incomplete studies. However, when the diagnosis has been deemed most likely, the treatment may consist of methods directed at lowering the intracranial pressure. Dehydration and repeated spinal-fluid drainage may in some cases control the disease. Advancing papilledema, with decrease in visual acuity and intractable headache, may make decompressive operative procedures imperative.

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ACUTE PORPHYRIA*

Report of a Case Showing Ineffectiveness of ACTH

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ALTHOUGH the metabolic disease, acute porphyria, has been known for many years, it remains a relatively unfamiliar syndrome to many physicians. Thus far, to our knowledge, there has been no report in the literature concerning the effect of ACTH on this condition.[1] In view of these features it was considered desirable to report a case of acute porphyria for which ACTH was administered.

Several excellent reviews[2-4] on porphyria have been published in recent years; accordingly, only a brief résumé of the essential features of this disease is recorded here. The congenital form, which may appear at birth or in early infancy, occurs more commonly in males; its clinical symptoms include such features as discoloration of the teeth caused by impregnated uroporphyrin, cutaneous lesions due to photosensitizing properties of the porphyrins and discoloration of the urine resulting from excretion of large amounts of these substances. Acute porphyria, which may be subdivided into idiopathic and toxic forms, occurs more commonly in females during the third or fourth decade of life. Photosensitivity is rare, and discoloration of the teeth absent. It is characterized by gastrointestinal and neuropsychiatric disorders. The patient experiences severe abdominal pains, either generalized or localized to the lower abdomen and pelvis. Nausea, vomiting and constipation are common. Involvement of the central nervous system may be manifested by a variety of neurologic abnormalities; however, an ascending (Landry) paralysis occurs most frequently. Psychiatric disturbances are also common. Menstrual disorders, especially amenorrhea, and cardiovascular involvement occur not infrequently. The diagnosis is suggested by these clinical manifestations, and may be confirmed by examination of the urine for porphyrins. The disease is actually a chronic condition, with numerous exacerbations and remissions, extending frequently over a period of many years. Acute toxic porphyria, a consequence of some toxic agent such as barbiturates, is clinically indistinguishable from the acute idiopathic form.

CASE REPORT

M. D., a 19-year-old married woman, was admitted to the hospital on April 14, 1949, because of severe abdominal pain and abnormal behavior. The family history was negative for neuropsychiatric abnormalities except for the occurrence of epilepsy in the paternal grandmother and excessive alcoholism in the mother. The patient's earlier medical history was noncontributory. Several unfortunate sexual experiences during her adolescence and late teens created marked sexual fears and conflicts. She was married at the age of 18, but sexual relations had been unsatisfactory. The menstrual cycle was normal. Constipation was present for 2 years before the present illness.

In March, about 1 week prior to the expected onset of menses, severe abdominal pain and exacerbation of the previous constipation developed. These symptoms gradually subsided with the onset of menses, only to reappear the following month, and on this occasion the symptoms continued after the menstrual period was established. She was admitted to a general hospital, where the diagnosis of acute appendicitis or oophoritis was entertained. Fortunately, however, abdominal exploration was not performed. Roentgenographic examination of the gastrointestinal tract was negative. During this hospitalization the patient was unco-operative; she refused nourishment, and intravenous administration of fluids was required. Further psychiatric abnormalities, including auditory hallucinations, threats of suicide and bizarre physical activity finally necessitated commitment to a mental institution.

FIGURE 1. *Appearance of the Patient.*

Physical examination revealed a poorly nourished female without other obvious significant physical abnormalities. The blood pressure was 118/94. Routine laboratory data were within normal limits. She continued to complain vigorously of lower abdominal pain, which bore no relation to eating or other physiologic activities. Because of hyperactivity, in-

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somnia, failure to eat and other disturbing symptoms, a course of electroshock therapy was administered, with satisfactory symptomatic improvement. She was permitted to leave the hospital in June, but returned in October because of severe abdominal pain, insomnia, restlessness and anxiety. After temporary improvement she was again released from the hospital in December, only to return within 6 weeks because of prominent physical debility and loss of weight. Again, she complained bitterly of pain in the lower abdomen and pelvis. At this time she was afebrile, but the white-cell count was 21,000, with 83 per cent neutrophils. One week later the count had declined to 15,400, with 82 per cent neutrophils, and in the following week these data were within normal limits. Other laboratory findings were normal. Within 2 or 3 weeks the abdominal symptoms subsided again, but at this point the patient complained of pains in the limbs. This was followed soon by a complete paralysis of the extremities and trunk muscles so that she was unable to sit up or turn over in bed. Incontinence of urine and feces appeared. Fortunately, there was no involvement of the respiratory muscles. Atrophy of muscles, particularly of the peripheral musculature, wrist drop and foot drop were prominent, being obvious even in photographs taken after the patient had exhibited some improvement (Fig. 1). Neurologic examination revealed, in addition to the above findings, loss of all tendon reflexes except for slight response by the left Achilles. Involvement of the cranial nerves, sensory system and cerebellar functions was absent. The neurologic picture and the history of previous repeated bouts of abdominal pain suggested the diagnosis of acute porphyria, which was confirmed by spectroscopic examination of the urine. Porphyrins were not present in the spinal fluid. During the succeeding several months the patient made slow but progressive improvement in her neurologic status. Excretion of porphyrins in the urine continued.

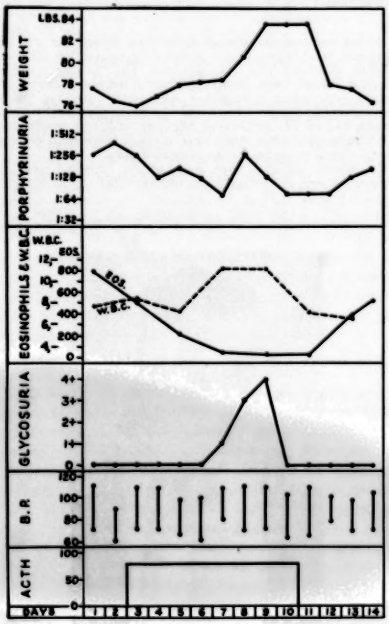

FIGURE 2. *Pertinent Laboratory and Clinical Data.*

In view of the absence of previous pertinent reports, it was decided to ascertain the effect of ACTH on this metabolic condition. Accordingly, 20 mg. of ACTH was administered every 6 hours for a period of eight days. During this time the patient indicated that her muscles seemed somewhat less tired than previously; otherwise, there were no significant objective or subjective changes. Laboratory and clinical data are indicated in Figure 2. There did not appear to be any significant effect on urinary excretion of porphyrins, which was approximately measured by dilution methods. This was also true during a later period, when ACTH was again administered. There were only minor changes in blood sugar, sodium, potassium and nonprotein nitrogen.

The patient was permitted to leave the hospital during the latter part of June since she appeared to be progressing satisfactorily. However, she returned within 3 weeks because of recurrence of severe abdominal pain and accompanying behavioristic disturbances. In order to observe its effect on acute abdominal symptoms, which had been absent during previous administration, ACTH was again administered in a dosage of 20 mg. every 6 hours during the first 5 days and 25 mg. every 6 hours during the following 5 days. There was no specific influence on the severity and course of the abdominal symptoms.

SUMMARY

A case of acute porphyria is described. ACTH did not appear to be effective in the treatment of this condition.

The technical assistance of Mr. Louis Faenza is gratefully acknowledged. Dr. Nicholas J. Giarmin, Department of Pharmacology, Yale University School of Medicine, performed the spectroscopic examination of the urine.

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THE FUNCTION OF A SUBURBAN HOSPITAL IN CIVIL DEFENSE*

The Concord Plan

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THE implementation of civil-defense training programs is not progressing with rapidity. Bradford's remarks on this subject[1] appear plausible. He stated, "The obstacle is the basic belief that it won't happen to us." One has but to recall Pearl Harbor to realize that it may happen at any time and in any place. That thought is being given to the problem, however, and progress is being made, are indicated in later reports.[2, 3]

Various estimates have been made of the number of casualties to be expected from an atomic-bomb attack. The number of bombs dropped upon a single city will determine largely the total number of casualties. The City of Boston may expect 500,000 casualties from each bomb dropped. Half this number will be killed.

These figures appear fantastic. Actually, they are not. They are based upon the effects of the atomic bombs dropped on Hiroshima and Nagasaki. Although the need for planning at national and state levels has been realized and implemented, the need for action at city and town levels has not. Planning alone at any level will not prevent utter chaos in case of an atomic-bomb attack. Teams of civilians, drilled in first aid, laboratory procedures, blood banking, intravenous therapy, treatment of burns and nursing care, must be ready in case of emergency. Preparations for such an emergency should be made now.

The Town of Concord, Massachusetts, has embarked upon such a training program. Spurred to action by the outbreak of hostilities in Korea, the staff of the Emerson Hospital drew up a list of objectives, obtained the approval of the trustees, appointed a representative to act and went to work.

The formal report was judiciously handled by the editors of the town's newspapers. Public support was immediate and gave strength for prompt follow-up action.

The objectives of the plan are summarized as follows:

Indoctrination of any group of citizens already organized for disaster control.

Co-ordination by physicians and public-health services for reviewing the preventive immunization status of the whole community.

Indoctrination of medical aides in large numbers, with emphasis on standard dressings for burns, splinting of fractures, treatment of shock and essential laboratory technics.

Training of a group in maintaining records.

Program of considered informative public education designed for controlling fear.

Development of a reference library on disaster planning.

Incorporation of dentists into the medical team.

Selection and further indoctrination of certain nurses in diagnostic screening, giving of transfusions, anesthesia and so forth.

Devise means of preparation, storage, and administration of food to burned patients in a practical manner.

An educational advising and co-ordinating committee was formed by the hospital staff, the trustees, the Red Cross, the local educators and the Co-ordinator of Civilian Defense. The committee members assumed the responsibility for recruiting class members, keeping in mind the qualifications they had deemed advisable.

The initial course in laboratory technic was followed by courses in intravenous administration of fluids, fractures and treatment of burns. These courses are being repeated and expanded. An experimental course in psychiatric technics, as related to civilian disaster, is now being given. Each course has been filled, and the waiting lists grow.

The Red Cross member of the educational advisory committee took immediate steps to start first-aid and home-nursing courses. Many members of these courses will be candidates for the medical-aid classes. Planning and co-ordination is such that duplication of instruction will be avoided to a large extent. The faculty for the medical-aid classes are free to experiment and have constantly modified their approach in keeping with the overall plan of the advisory committee. The original plan to have teen-age medical aides has been modified to include parents with daughters or sons and educator-student teams.

Plans for an extension course in laboratory technics for a group of officers and inmates in the Concord Reformatory in preparation for a blood-banking center at the farm building are being carried out. Original plans for evacuation of the Middlesex School of teen-agers to their homes in other parts of the country in the event of disaster have been modified by the school physician. The students of

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the Middlesex School will participate in medical-aid training to be given by their physician, with the co-operation of other members of the Emerson Hospital faculty, thus building up a strong corps of hospitalmen.

The addition of the Reformatory Farm, as a blood-banking center, and the Middlesex School, as an evacuation hospital, to the facilities of the Emerson Hospital called for more trained medical personnel than Concord could supply. This need has been met by a volunteer group of Inactive Naval Reserve Medical Department personnel, composed of 60 Medical Corps officers, 20 Nurse Corps officers, 6 Medical Service Corps officers, and 40 trained Hospital Corps men, who have been assigned to volunteer duty with the Emerson Hospital and its allied facilities. This group will respond upon hearing the announcement of a major disaster to the Boston area over public communication systems. The Naval reservists, the Emerson Hospital staff, and the civilian teams in the process of training will constitute a well organized unit that can cope with a great number of casualties.

To date, the various civilian groups have studied blood counts, intravenous technic, care of fractures, burns and first-aid principles. Participants in the course in intravenous therapy have drawn blood from each other. In the laboratory section, accurate white-cell counts have been performed. Gradually, the feeling of fear and uncertainty has given way to one of assurance and competence.

Should a disaster occur in the Boston area today, Concord stands ready to meet the emergency with a fair chance of avoiding major confusion. As the training continues, the prospect grows of a smoothly functioning organization that can be adapted to any over-all plan.

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FIFTY YEARS OF MEDICAL PROGRESS

MEDICINE AS A SCIENCE: PEDIATRICS

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A REPORT on pediatrics for the first half of this century is, in fact, the record of the birth of a specialty in medicine and its growth to maturity. In 1900 there were a very few men devoting themselves exclusively to the care of infants and children, and probably not over 50 medical practitioners in the whole country who took a particular interest in this age group. Possibly 1 doctor out of every 2500 in the United States could have been classified as a pediatrician, if the definition were interpreted very liberally. These men were all located in the large cities, and nearly all of them saw adult patients as well as children. Today there are over 3000 doctors certified by the Board of Pediatrics, or about 1 in every 60 of all registered physicians. It is significant, also, that of the approved residencies available in the United States about 1 in 18 is in the field of pediatrics, and all but 8 of the available residencies were filled at the time of the last American Medical Association Report.

This development of pediatrics as a specialty has been accompanied by a change in pediatric education. Like practice, teaching in relation to infants and children was a part of the instruction in the courses of general medicine. Gradually, certain teachers began to take a special interest in the younger age groups. Abraham Jacobi was a pioneer in this movement. In 1857 he began teaching at the bedside and continued to devote himself to the problems of infants and children until his death in 1919. In 1861, Job Lewis Smith was appointed clinical professor of the diseases of children at Bellevue Hospital in New York. In 1888 Thomas Morgan Rotch was appointed assistant professor at Harvard Medical School and, in 1893, full professor with a chair on the faculty. This was the first real professorship of pediatrics in this country except for an appointment of short duration at Boston University in 1890. L. Emmet Holt was appointed professor of diseases of children in the New York Polyclinic and, in 1902, at Columbia.

Up to the early part of this century, persons wishing to pursue special work in pediatrics went abroad, particularly to Germany, to study under Czerny and others. In 1912 the first full-time pediatric clinic in the country was established at Johns Hopkins, and John Howland was appointed as the chief. In 1908 von Pirquet had been appointed professor of pediatrics at Hopkins, but he remained only two

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years. Following the example at Baltimore, pediatric clinics spread rapidly in the next ten years. At the present time every medical school recognizes pediatrics as one of its major departments. Coincident with the growth of pediatrics departments in medical schools has been the establishment of pediatric wards in most of the large general hospitals and a few special hospitals for children. As the specialty of pediatrics grew in stature it became apparent that surgical procedures in children differed radically from those in adults. Also, many congenital malformations presented a challenge for correction, which was ignored by most general surgeons. Certain surgeons, however, in various parts of the country, began to take a special interest in infants and children and to appreciate their peculiar problems. William E. Ladd, at the Children's Hospital in Boston, soon became the recognized leader of this group and through his inspiration stimulated the rapid recognition of infants and children as a field for special surgery. In 1939 there was established at Harvard Medical School the William E. Ladd Professorship of Child Surgery, the first such chair in the world. The differences in the manifestation and course of disease in children from that in adults led to sub-specialties in pediatrics. At the present time there are many persons devoting themselves entirely to pediatric allergy, cardiology, psychiatry and other fields.

Pediatric societies came into existence at the end of the last century, indicating the increasing awareness of the special problems of infants and children. The Section of Diseases of Children of the American Medical Association was organized in 1880, the American Pediatric Society, in 1888, the Academy of Pediatrics, in 1930, and the Society for Pediatric Research, in 1929. The American Board of Pediatrics was established in 1933. The Academy of Pediatrics now has over 2500 members and, in addition to its contribution to scientific medicine, exerts a tremendous influence upon all community activities relating to children. Its recent study of child-health services in this country is the most comprehensive survey ever made by a medical organization, and its findings will form the basis for work in this field for many years.

Titles dealing with children have been in the medical journals from their earliest editions. These increased rapidly in the late 1800's, and a few special pediatric journals began to appear, mostly in Germany. In 1884 the *Archives of Pediatrics* was published in this country, but no other special pediatric journal appeared until, in 1911, the American Medical Association established the *American Journal of Diseases of Children*. In 1926 the *Journal of Pediatrics* was published under the aegis of the Academy of Pediatrics, but it is now entirely independent. In 1948 the Academy published its own journal, *Pediatrics*. There are also published reviews of pediatric literature and extensive studies in the field of growth and development. The pediatric titles in general journals have increased to a very large number. Pediatric textbooks have also been published in standard editions and in loose-leaf bindings.

Pediatrics differs from all other specialties in that it does not confine its interest to a particular system of the body or to a single disease. The pediatrician is concerned with the whole person, as is the internist, — his physical, mental, emotional and environmental problems, — and limits his activities only by excluding the care of patients beyond the age of maturity. There has been a striking increase in interest in the growth and development of the fetus to the mature adult during the last fifty years. This has come in the laboratory, in the clinic and in the field, and much of it from outside medicine. Whether the growth of pediatrics as a specialty has been the stimulating spark for the study of growth and development, or whether the interest in growth and development has stimulated the growth of the pediatric specialty is beside the point. The two have gone forward simultaneously, and the importance of the subject and the recognition of the peculiar problems presented by changing bodily structure and function have established pediatrics on a firm basis. The pediatrician is no longer considered merely a "baby feeder" as he was fifty years ago, but has taken his place as a respected teacher, investigator and practitioner in the medical fraternity.

Studies in nutrition have been pursued in many clinics, and much knowledge has come also from the laboratories of pure science. It has been shown how important the complex details of diet are in influencing growth and development. The pediatrician has been able to utilize much of this new knowledge in the practical feeding of infants and children. He knows something about the individual food elements and their functions in nutrition, of the mineral constituents in the diet and particularly the role of vitamins, whose existence was not known at the beginning of the century. He no longer sees marasmic infants, and scurvy and rickets have almost disappeared as diseases of infancy. From recent studies it appears that the present-day mature child is taller and heavier than his grandparents were at his age.

Coming out of the studies of growth and development has been the setting up of normal standards for the various age groups. At first these consisted largely in measurements of height and weight. The tables prepared by Baldwin and by Wood were in constant use for many years and have not now been entirely discarded. It was recognized however that other measurements were needed if a true picture of growth and development were to be presented. Rotch was the first to call attention to the epiphyses, as studied by x-rays, as indicators of the state of bodily maturity. From this followed the work

of Todd and others in this field. It would require more space than this report is allotted to mention the many persons who have made important contributions to normal standards in gross body structure and in chemical and microscopical composition. The meticulous measurement of basal metabolic rates by Fritz B. Talbot furnished the first factual data on infants in this phase of bodily function.

In the realm of mental growth and development the outstanding observations of Gesell must be recorded. Much credit goes to Aldrich for emphasizing the importance of emotional factors. The general increase in interest in psychiatry is of special significance to pediatrics, for it is in the younger age groups that the hope of prevention lies.

All the early attempts to establish normal values were based on studies of groups of infants and children. These were obviously open to criticism; more accurate values could be obtained by longitudinal studies in which the same groups of children were investigated as they progressed from birth to maturity. It was also possible in such groups to gain some knowledge of parental factors and the course of pregnancy. The work of Washburn and of Stuart is of outstanding merit in obtaining data of true significance.

Pediatricians from the first were concerned with the infant, and especially with the problems of feeding. The newborn infant, both full term and premature, has received increasing attention as time has passed. The fact that most infants are born in hospitals, and that in most of these the care of the infant is turned over to the pediatrician immediately after birth, has made possible opportunities for study, and real advances in knowledge have resulted. The close co-operation of the obstetrician has been of importance in many matters: the type of anesthesia given to the mothers, time of labor and technics of delivery. It has been shown conclusively that many maternal conditions do influence the health of the infant. The early reports of Gregg, from Australia, on the effects on the fetus of German measles in the mother during the first trimester of pregnancy have been fully verified. The work of Warkany and others indicates that in animals, at least, nutritive deficiencies in the mother may result in malformations in the offspring. The careful correlation of the stages of fetal development with specific defects in the infant has added new stimulus to the study of congenital malformations. The work of Ingalls on mongolism is an illustration of the value of such studies. Blood groups were discovered early in the century but it was not until 1941 that Levine demonstrated the importance of agglutinins in the highly fetal icterus gravis of the newborn. Erythroblastosis fetalis is now a well recognized disease amenable to treatment. Studies of the physiology of the fetus and the newborn have been increasingly productive. Many persons have made contributions in this field. Clement A. Smith's book is the most comprehensive publication on the newborn since Van Rues's classic work, published in 1914, and has become the standard textbook on the subject.

The feeding of the infant, both full term and premature, has undergone marked changes since 1900. Everything known about vitamins and most of what is known about the food elements and minerals, so far as they affect growth and development, has been acquired in this time. The pediatrician has had to modify his ideas and methods of feeding to conform to this new knowledge. Breast feeding still remains the best method of nourishing the newborn infant. Its popularity waxes and wanes. Just now it waxes. Its waning was due primarily to the improved methods of artificial feeding and the demonstration that the former high death rate among infants not fed at the breast was due to factors that were largely amenable to control.

Infants whose mothers did not nurse their babies were given, fifty years ago, either milk from some other woman (and this rarely) or a poorly devised concoction of which cow's milk was usually the basis. The milk was dirty — usually not being sterilized — and was put into dirty bottles and fed through dirty nipples. Proprietary foods, usually deficient in most elements except carbohydrates, became popular. Then came the movement for clean milk and studies of its composition, followed by the period of modified milk feeding — an effort to simulate breast milk and to find the best food for the infant. In view of present-day knowledge the formulas then in use seem poorly to meet the requirements of a proper diet, but they did mark a real advance over former methods of feeding, and indicated the search for a food that would meet the needs of the growing infant. The troubles of that period were due in part to the failure to differentiate, in the infant who was doing poorly, the role played by infection as against food inadequacies. Owing to the better understanding of nutrition, infants are now given an artificial feeding with supplementary vitamins, which is safe, and supplies, so far as they are known, the requirements for growth and development. This does not mean that knowledge is complete. New facts, as they come to light, will further modify the practice, but at least the basis of infant feeding today is scientific knowledge and not ignorant guesswork.

The psychologic factors concerned in feeding have received increasing recognition in recent years. It is realized that emotional reactions are operative from birth and should be given consideration. "Rooming-in," in which the newborn baby is kept in the same hospital room with the mother, "demand feeding," in which the infant is fed when he asks for it, and similar practices are in line with these ideas.

Prematurity still remains the most important cause of death in the first few days after birth. Special nurseries for premature infants are now accepted as essentials in lying-in hospitals. The Chapple bed and the Isolet have made possible isolation of the premature infant, even in a general nursery. Careful studies by Levin and Gordon and others have influenced the feeding of premature infants to a marked degree. Real advance in the care of the premature baby has been made, but much remains to be accomplished. Retrolental fibroplasia, first reported by Terry and Clifford, is an unsolved problem both in etiology and in treatment. The value of community planning for the premature infant has been demonstrated by Julius Hess and his work in Chicago. State and federal funds are now expended in large amounts to implement such community plans. The influence of the Children's Bureau and the work of Ethel Dunham deserve special mention. The Academy of Pediatrics, through the committee of which Stewart H. Clifford is chairman, has made available a brochure containing the most recent opinion and practice in relation to the premature infant, and is doing a great service in improving the care of the premature infant all over the country. Since the pediatrician is interested in the whole period of life during which growth and development take place, it is natural that he should be concerned with adolescence. This late period before maturity has been neglected and has only recently received even passing attention. Studies are now going forward in relation to the physiologic changes that occur and to emotional and psychologic considerations. Educational and vocational guidance is also involved. Thus far only a beginning has been made in an understanding of this age group.

Pediatrics has benefited by progress in the whole field of medicine. This is strikingly true of the research in specific diseases and the improvement in treatment that has resulted. A considerable body of knowledge about methods of prevention of certain diseases has now accumulated, and, since the morbidity and mortality of many of these diseases are particularly high in the younger age groups, pediatricians have adopted preventive practices with great enthusiasm and with striking results. Diphtheria is now a rare disease in childhood since the early work of Schick, in Germany, and of Park in this country, in developing toxin-antitoxin, and the later introduction of diphtheria toxoid. Pertussis is still an important disease, but its incidence in the early years of life is much less than before the widespread use of vaccines, following evidence from Sauer's work of their effectiveness in preventing or alleviating the disease. The use of antibiotics in treatment has further reduced the severity and mortality of this disease.

Smallpox vaccination, although not new during the last half century, is now almost universally employed. The demonstration that the occasional complication of encephalitis after vaccination practically never occurs, if the primary vaccination is done before eighteen months of age, has resulted in early vaccination and thus early protection. Tetanus toxoid is now given routinely to infants during the first year of life. The production of preparations containing multiple biologic products has been of great advantage to immunologic practice. McKhann, in 1933, demonstrated that an extract from the human placenta if given to persons exposed to measles would prevent or reduce the severity of the disease. During World War II, Cohn, in the Laboratories of Physical Chemistry at Harvard University, developed methods of fractionating blood. From this source serum globulin was made available for experimental studies. It is clear now that the value of placental extract was due to its globulin content. The work of Janeway and of Stokes showed clearly the effectiveness of gamma globulin in measles and opened a wide vista for study.

Meningitis has been successfully treated, first by serum for the meningococcus, produced by Flexner in the Rockefeller Institute, in the second decade of the century, and later by the antibiotics and serum for influenza, produced by Hattie Alexander. Pneumonia was formerly a very fatal disease in infants, but, like many other infections, it has yielded to specific drug therapy. Scarlet fever has been robbed of its terrors by the use of antibiotics.

The virtual disappearance of so-called summer diarrhea in infants, which took a tremendous toll of lives in 1900, is one of the triumphs of preventive medicine. Dysentery, or infectious diarrhea, has also largely disappeared as an important disease of early life. These changes have been due to many factors, primary among which are the increase in knowledge of nutritive requirements, the pasteurization of milk, the child-health movement and better treatment of those who are sick. The last factor is due in large measure to an understanding of the importance of fluid and electrolyte balance, gained from the early work of Gamble and others working in Howland's laboratory, and later by the work of Marriot, Hartman, Darrow and many others. Also, a real contribution has been made by the use of blood transfusions and other supportive measures.

One might continue at great length this list of diseases that have been reduced either in incidence or in mortality. These changes have been due in some instances to sanitary control, in others, to the discovery of etiologic agents and specific therapy, and in still others to improvement in general therapeutic procedures. These advantageous changes in the disease picture have benefited all age groups, but the high morbidity and mortality of many in-

fections in children have had a striking influence in pediatrics.

Preventive medicine has found its most consistent support from pediatricians. Very early in medical history interest was manifested in the well child, and measures were adopted to maintain his health. In the last century, more organized efforts appeared — particularly in France — centered for the most part around the newborn infant and his feeding and care during the first year of life. In this country the experiences of Europe were repeated, in that the efforts for better infant care were activated by individuals or small voluntary groups, most often under nonmedical sponsorship. In 1889 Koplik introduced the distribution of modified-milk formula for infants from stations in New York City, and three years later Nathan Strauss provided pasteurized milk free to many babies. Coit introduced certified milk in 1891, and, although it has never been used extensively, it has exerted a great influence in raising the standard of milk employed in infant feeding. Milk stations soon were established in many large cities. At them mothers were given advice about the care and feeding of their infants. Gradually, the sphere of interest widened and they became the modern child health centers, where every aspect of the child is considered. They have in many places been linked closely with prenatal clinics — evidencing the intimate association of maternal health and the health of the infant.

In association with these centers there has been a rapid advance in the work of the visiting nurse, supplementing and interpreting the advice of the doctor given at the clinic. At the beginning of the century infant mortality in this country was very high despite the efforts of these individuals and groups. In 1909 the American Academy of Medicine sponsored the first conference on the subject in the United States. The conference met at New Haven with Dr. Helen C. Putnam as chairman. Out of the conference grew the organization of the American Association for Study and Prevention of Infant Mortality, with J. H. Mason Knox as its first president. This association immediately took the lead in developing the child-health movement, which is one of the great achievements of this century so far as work with children is concerned. It attracted the foremost pediatricians in the country, who have worked together since that time in furthering all the activities, both voluntary and governmental, having to do with children.

It would be impossible to allocate to individual physicians their particular contributions to the combined effort. Samuel M. Hamill and Philip VanIngen should receive special mention, for they both worked unceasingly in every phase of the many activities and inspired others with their great unselfishness and wise counsel. Through the work of this first organization and its successors, and that of other organizations with similar objectives, there has been a great reduction in infant mortality all over the country. Exact figures for 1899 are not available for the United States as a whole since the birth registration area was not established until 1915. Statistics for Massachusetts are typical of the northeastern section. The infant mortality in Massachusetts in 1899 was 149.5, and in 1949, 24.4. Child-health stations have been established all over the country. Mothers have been taught better care of their babies. The preschool child has also been included in the sphere of interest, and much has been done to improve his physical health, and some assistance given parents in guiding his emotional life and his adjustments to community life. Stimulation has been given to the widespread adoption of immunologic procedures.

In 1920 this first association changed its name to the American Child Health Association, in recognition of its expanded program of interest in the child from birth to maturity. In 1923 it combined with the Child Health Organization, which had been formed five years earlier and which was concerned primarily with health education, to form the American Child Health Association, of which Mr. Hoover was the first president. This association was dissolved in 1935 because, as such, it was no longer needed. The work was being done by other professional and governmental groups.

In 1910 President Theodore Roosevelt called together the first White House Conference on Children. Since then such conferences have been called by the president every ten years. Out of these conferences have come many of the ideas resulting in improved care of infants and children — especially that of 1930, the publications from which are still the authoritative texts for instruction in many phases of child health and welfare, primarily the fundamental data on growth and development. The slow utilization of many of these data illustrates how far practice falls behind in applying known knowledge. That the 1930 conference is still a potent factor in influencing work with children should encourage those who look for quick results and are disappointed in accomplishments.

In 1912, by Act of Congress, the Children's Bureau was established in the Department of Labor and later transferred to the Social Security Agency. This grew out of the first White House Conference and was suggested to Theodore Roosevelt by Lillian D. Wald. Miss Julia Lathrop was appointed its first chief, and great credit is due her for inspiring leadership and sound planning. She was succeeded by Miss Grace Abbott and by Miss Katharine Lenroot. Up to the creation of the Children's Bureau, the federal Government had manifested some interest in children through the work of the Public Health Service but, for the most part, it was concerned with collecting statistical data and in quarantine regulations. The Children's Bureau immediately gave impetus to the establishment of child-

health divisions in state health departments, to improving birth registration and vital statistics and to fostering prenatal care and infant-welfare activities by governmental and private agencies. From 1921 to 1929 it administered the Sheppard–Towner Act, the first effective act in the United States for the protection of women and children. Increasing authority and appropriations have been voted to the Bureau as the federal Government has expanded its interest in child health. Today in almost every phase of child care the influence of the Bureau is felt, and many advances have been possible because of funds allocated by it through state health departments or to private organizations.

In 1908 the first bureau of child-health in the United States was established in New York City, and Dr. Josephine Baker appointed as director. The example of New York City was followed quickly in other parts of the country — in no small measure because Dr. Baker did such a good job in New York. Today, every state, as well as most large cities, has a child-health division in the department of health.

It has been evident from the above statements that interest in children in the beginning was centered around the infant, but the preschool and school child are now receiving equal consideration. In 1899 the first official action for the health of the school child was taken in Boston in the setting up of regulations for the exclusion from school of children ill with contagious disease. Today, governmental agencies, medical societies and many lay groups are exerting combined efforts to improve the health of school children and to ensure the optimum environment, physical and personal, during their school life. In the educational field the work of Terman should be mentioned. He was one of the first educators to appreciate the importance of the health of children in the schools. Now most private and public schools and colleges have active departments of health, which are concerned with all phases of school life that bear upon the health of the scholar. Child labor laws have been enacted to prevent the exploitation of children in industry, agriculture and other fields.

In this growth of the child-health movement it is apparent that the Government is taking an increasing share in financing and planning. Through the leadership of the pediatric societies — especially the Academy — the execution of plans and the determination of policies and procedures are in the hands of pediatricians. The recent study by the Academy of child-health services in this country clearly indicates that, although great advances in child care have been made in the last fifty years, much more needs to be done, particularly in making available to all children the facilities now known to be valuable but denied them by reason of economic or geographic circumstance.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

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CASE 37051

PRESENTATION OF CASE

A seventy-two-year-old man entered the hospital because of vomiting of twenty-four hours' duration.

The patient was quite ill and somnolent, and the history was obtained chiefly from his wife. Six months before entry he began to notice increasing difficulty in bowel movements, but there were no bloody or tarry stools and no change in their caliber. There had been no significant weight loss. Six days before admission his obstipation became more severe, and he had not evacuated his bowels for five days preceding entry. Fatigue and anorexia ensued, and for the two days before entry he passed no gas whatever. Twenty-four hours before admission he began to vomit brown, watery, malodorous material; this persisted up to the time of admission.

He had had influenza in 1918, and pneumonia in 1921 and 1939. Surgical amputation of the nose had been done in 1946 because of squamous-cell carcinoma. He had suffered from progressive paralysis agitans for the past ten years. In recent visits to the Out Patient Department he had complained of pain in both groins radiating to the penis, which he said at that time had been present for two years, but abdominal examination had been negative. On admission, however, the groin pain was denied, and the patient and his wife said that they had noticed a small lump in the right groin for one year.

Physical examination showed a thin, dehydrated, drowsy, acutely ill man, with marked Parkinsonian tremor. The nose was absent. The heart and lungs were not abnormal for his age. The abdomen was slightly distended (particularly in its lower portion) and tympanitic, with mild diffuse discomfort on

pressure. Occasional high-pitched peristaltic sounds could be heard. No masses or organs could be felt. A small, firm, nontender, slightly irregular, irreducible subcutaneous mass 2 by 2 cm. was present in the right groin, overlying the inguinal ligament medially to the femoral pulsation. Rectal examination was negative.

The temperature was 100°F., and the pulse 90. The blood pressure was 130 systolic, 70 diastolic.

Urinalysis was negative. A stool guaiac test was negative. The white-cell count was initially 18,000; after six hours of hydration it fell to 7000, and thereafter ranged from 4700 to 7000. The hemoglobin was 12.0 gm., which subsequently rose to 14.7 gm. after several transfusions. Plain x-ray films of the abdomen showed gas-distended loops of small bowel in the upper and lower left abdomen. Much feces and a small amount of scattered gas were found throughout what was thought to be the large bowel. In a chest film, linear streaking densities were seen in the midportion of the right lung base, perhaps representing bronchiectatic changes. The cardiac silhouette was normal.

A Miller–Abbott tube was passed, and parenteral fluids were begun. The patient was started on penicillin and streptomycin. On the following morning the nonprotein nitrogen was 86 mg. per 100 cc., and the chloride was 86 milliequiv., the sodium 133.0 milliequiv., the potassium 4.5 milliequiv. and the carbon dioxide 36.3 milliequiv. per liter. A barium-enema examination without preparation revealed no gross obstruction in the large bowel.

For the first few days fluid therapy did not prevent a further fall in chloride, although it rapidly brought the nonprotein nitrogen down to 31 mg. per 100 cc.; subsequently the chloride rose to 97 milliequiv. per liter. During this time the patient grew more somnolent and could be aroused only with effort. The abdominal distention slowly responded to intestinal suction, but the patient's general condition remained poor. High colonic irrigations were carried out daily in an attempt to evacuate residual feces and barium from the large bowel. On the seventh hospital day the intestinal tube was well down in the pelvis, though several loops of gas-filled small bowel were still present. On the eighth hospital day, several observers were unable to palpate the right groin mass, but on the following day the mass was again present and still nontender. At this time his general condition was better than at any time since admission, he was more alert, and he responded to questions. He exhibited for the first time slight tenderness to palpation in the lower abdomen, with voluntary spasm, which subsided two hours later. An operation was performed.

Differential Diagnosis

Dr. Claude E. Welch*: I have found that many of the histories obtained from very sick patients or from their wives are not necessarily accurate. Therefore, we do not have to pay too much attention to the part of this patient's history that suggests carcinoma of the colon. Nor do I think that the disease was a sequela of the carcinoma of the nose. That would be very bizarre indeed.

Undoubtedly, at the time the man was first examined, it was decided that he had intestinal obstruction, presumably due to an increased femoral hernia. The story is consistent with that. The question of strangulation of the hernia would immediately arise, particularly with the mild diffuse discomfort on pressure in the lower abdomen, which is a sign of interference with the blood supply, or of inflammation, rather than of uncomplicated small-bowel obstruction. The fact that there were occasional peristaltic sounds gave the physicians enough confidence to proceed with a more detailed examination and with conservative therapy instead of rushing the patient to the operating room.

What was the temperature chart during the course of the illness?

Dr. Benjamin Castleman: The temperature was elevated when he came in, but quickly became normal and remained so.

Dr. Welch: May I see the x-ray films?

Dr. Stanley M. Wyman: The first film shows distended gas-containing loops of small bowel, measuring perhaps 3.5 to 4.0 cm. in diameter. The colon contains retained material and very little gas. Only small segments of it can be identified. There is some indentation of this portion of the ascending colon, but I can draw no definite conclusions from this.

Dr. Welch: Does this shadow suggest the edge of a mass, with gas pushed over in this direction?

Dr. Wyman: It could perfectly well be a mass. The chest film shows slight accentuation of the markings in the lower lung fields, perhaps chronic bronchiectasis, but nothing else remarkable.

The colon was not prepared for the barium-enema examination. The barium flowed throughout the colon into the cecum, showing no gross obstruction; I can make no definite statement about it. There is, however, some pressure on the right colon, but I am unable to attach any great significance to it.

Dr. Welch: There is no real question about the immediate diagnosis in this particular patient — acute small-bowel obstruction. The problem is to guess at the probable cause, because I find it very difficult to arrive at a definite diagnosis. Instead of going over one or two dozen different causes of obstruction, I think it might be desirable to consider merely three of the more likely ones and to hope that the diagnosis may fall into this group.

I think I can assume that the primary lesion was in the lower abdomen, and that the primary source of the obstruction was in the small bowel, the colon or the appendix. Let us consider first acute small-bowel obstruction arising from some lesion in the small bowel itself. Statistically, almost 45 per cent

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of all small-bowel obstructions are caused by hernias; and this man had a lump in the groin quite consistent with a hernia — particularly of a femoral type. I suspect that the man's diagnosis, when he was treated in the hospital, was that of a partial obstruction due to Richter's hernia, which is an incomplete hernia through the femoral ring. That is not an unattractive diagnosis, but several features are against it. One is that this man was sick for about six days before he came into the hospital. With an incarcerated femoral hernia, the patient usually becomes very sick very rapidly, there is little question about the diagnosis of intestinal obstruction, and unless operation is done rapidly there is likely to be perforation of the bowel. Furthermore, it is a little unusual that this man took about a week, with an obstruction of the small bowel, to pass a Miller–Abbott tube down to the pelvis. That would usually be done much more quickly, unless the man had some element of paralytic obstruction. The fact that the hernia went away and came back again is against the diagnosis of obstruction caused by Richter's hernia. When the hernia disappeared, undoubtedly the examiners were concerned with the possibility of a spontaneous reduction en bloc of a hernia, together with its incarcerating ring. Finally, hernias are so common in old people that I am inclined to regard them as "red herrings," rather than as the original causes of obstruction.

To continue with the small-bowel lesions, could there be something else, such as a band or a venous thrombosis of the distal ileum? There is no evidence in favor of either condition, and I merely mention them in passing. Suppose the primary lesion were in the colon, and there was an overlooked carcinoma with metastases in the cul-de-sac producing small-intestine obstruction rather than obstruction of the large intestine. Or perhaps the small intestine was adherent to a diverticulum, which produced small-bowel obstruction. They are very remote possibilities. The one thing that suggested them was the rather hazy history that was taken when the patient first came into the hospital. I do not believe the primary lesion will be found in the colon.

Could the whole picture be explained by acute appendicitis, with secondary intestinal obstruction? I must say that when I went over this protocol, I was impressed with that possibility. I am more than disappointed by the fact that I can see the proximal portion of the appendix on the x-ray picture. Appendicitis in an old patient may produce a picture that is very difficult to diagnose. There is usually much more disease inside old people than is usually believed when they are first seen; and that is doubly true with appendicitis. Most of this picture can be explained on the basis of appendicitis. The patient was sick for about six days before he came in, and finally he developed signs of intestinal obstruction, indicating that there had been a progression of the primary lesion over the course of six days. The tenderness noted when he first came in was also suggestive. The slight tenderness to palpation of the lower abdomen, with voluntary spasm, which he demonstrated just before he went to the operating room, is again evidence of an inflammatory lesion in the right lower quadrant, of which the appendix would be the most likely source. That the man remained a little toxic despite the fact that the intestinal obstruction was relieved also might be adduced as further evidence in favor of it.

I shall say that the man had acute small-bowel obstruction, that he had an inflammatory lesion in the right lower quadrant that produced it, that the hernia was not the cause of the obstruction and that probably the cause of the inflammatory lesion was acute appendicitis.

Dr. Allen G. Brailey: You are aware of the story of increasing constipation over six months?

Dr. Welch: I am; I think that was obtained under duress.

Dr. Wyman: What about the white count?

Dr. Welch: A count of 18,000 on entry, going down to 4700, does not help me one way or the other.

Dr. Castleman: Would you tell us the opinion on the ward, Dr. Newstedt?

Dr. Roger Newstedt: For the most part, our reasoning followed the same pattern that Dr. Welch used. When the patient first came in he was quite sick, and with the 18,000 white-cell count and the temperature of 100°F., we anticipated that he would need considerable urgent treatment before he would be fit for surgery. He had been vomiting for a full day. To our surprise, on the following morning after only six hours of treatment the temperature was normal and the white-cell count had gone down to 7000 — yet the fluid treatment had been inadequate; so that we did not feel we had been right in assuming he had peritoneal irritation in addition to the obstruction. He developed over that time definite signs of increased somnolence and toxicity, which led us to believe that he was in no condition for surgical treatment. We had a barium enema done, so that we could be sure the large bowel was not involved. We proceeded on the assumption that he had small-bowel obstruction in addition to the hernia. We believed that the mass in the right groin was an incarcerated femoral herniation that had been there for years without symptoms. As time went on, and he continued to have a normal temperature and white-cell count, we assumed that he did not have strangulation obstruction. Part of the delay in operation was due to the time required to evacuate the barium. Therefore we thought that he probably had omentum in the hernia that was acting as a fixed point, and that allowed the small bowel to be obstructed around it without endangering the blood supply to it. Our preoperative diagnosis was small-bowel obstruction with femoral hernia,

and probably obstruction due to an adhesive band, or something of that sort. We considered Richter's hernia, but we believed we had eliminated that because of the laboratory and abdominal examinations.

Clinical Diagnoses

Obstruction of small bowel due to adhesive band or torsion.
Hernia, femoral.

Dr. Welch's Diagnoses

Small-bowel obstruction, due to acute appendicitis.
Femoral hernia.

Anatomical Diagnosis

Femoral hernia with partial incarceration of small bowel (Richter's hernia).

Pathological Discussion

Dr. Newstedt: At operation he did have a femoral hernia, but he did not have omentum in the hernia. A button of small bowel protruded from the antimesenteric border of the ileum about eighteen

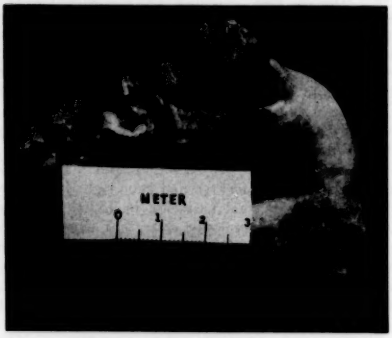

Figure 1.

inches above the ileocecal valve. The bowel itself was acutely angulated, so that nothing was going through it, but the blood supply was not cut off. To us, it looked like either Richter's hernia or possibly Meckel's diverticulum.

Dr. Castleman: Here is the small intestine, and here is the button-like area that was herniated into the femoral ring (Fig. 1). It was very small. If this were a Meckel's diverticulum, it would be one of the shortest we have seen. On opening the bowel, we found that the rugae of the bowel continued along in the herniated portion, so that we felt it was not a Meckel's diverticulum; and on microscopical examination there was no stomach epithelium, although this does not rule it out. This was really a small portion of the bowel, knuckled into the femoral ring, which showed healing inflammatory reaction and no real gangrene. Undoubtedly this was the cause of the small-bowel obstruction.

Dr. Welch: I assume the mass must have been there on the day the examiner did not find it.

Dr. Newstedt: It is possible that it was there, and that we missed it. The patient was examined in the lateral position. Dr. Irad Hardy and I felt for it and did not identify it. At the time of operation, after dissecting it out, we found that the whole mass would reduce en bloc, and we had to cut the peritoneal ring, in addition to the inguinal ligament, to reduce it; so it is still possible that it reduced en bloc on that particular day. All things considered, we believe that we missed it.

CASE 37052

Presentation of Case

First admission. A fifty-five-year-old man was admitted to the Massachusetts Eye and Ear Infirmary because of redness and pain in the eyes for six months.

The condition had had a gradual onset, increasing in severity in spite of local medication of various types. Roentgenograms had revealed a pansinusitis, particularly of the left antrum. This had been drained three times in the three weeks prior to admission. He had not noticed any impairment of vision, and had no other complaints.

The family history was noncontributory. The past history included a hemorrhoidectomy and excision of a fistula in ano one year prior to admission, and excision of three small masses of unknown nature from the left buttock six months prior to admission. All his teeth had been extracted eight months prior to admission, because many had been carious. Although the right leg and arm had been somewhat shorter than the left for as long as he could remember, he had been accepted for military service in World War I. However, he had been

discharged because of hip pain associated with military duty.

Physical examination revealed a well developed, well nourished man, with the right leg 4.0 cm. shorter than the left and the right arm 2.0 cm. shorter than the left. The heart and lungs were normal. The mucous membranes of the nose were red, but there was no pus. There was beefy-red episcleral injection of both eyes. The right eye showed a tension of 32 and the left, 34. The pupils measured 0.25 cm. and did not react. The globes were slightly tender. There was little or no real discharge.

The temperature, pulse and respirations were normal. The blood pressure was 150 systolic, 90 diastolic.

The urine had a specific gravity of 1.002, and contained no sugar or albumin. There were occasional white cells in the sediment, but no red cells or casts. Examination of the blood disclosed a hemoglobin of 11.8 gm., a red-cell count of 3,960,000 and a white-cell count of 9100, with a normal differential. Culture of the nasal discharge revealed alpha-hemolytic streptococcus and *E. coli*. Cultures from the eyes revealed *B. subtilis* on admission, and no growth on two subsequent cultures. A roentgenogram of the chest was normal. Roentgenograms of the sinuses revealed pus in the left frontal sinus and the antrum.

Treatment in the hospital included local applications to the eyes of 4 per cent pilocarpine, mecholyl, carcholin, epinephrine bitartrate, and furmethide. There was no perceptible change in the tension or in the redness. The sinusitis was treated with penicillin, aureomycin, streptomycin, sulfadiazine (orally), Cortisone drops and antral washes, with apparent clearing of the sinusitis. The temperature ranged from 98.6 to 100.2°F. He was discharged after nineteen days in the hospital, to continue further treatment at home.

Final admission (six weeks later). The patient was readmitted on the Medical Service because of chills, fever and malaise.

He had stayed at home after discharge from the hospital, and had noted little change in the eye condition. Two weeks prior to admission he had a shaking chill that lasted fifteen minutes. During the week following this he felt weak and "feverish." He noted a transient swelling of the right ankle lasting only a day or two. His doctor confirmed the presence of swelling and also stated that the ankle was red. One week prior to admission he had a second shaking chill, following which the temperature rose to 102°F. He was given streptomycin tablets by his local physician, and remained in bed until admission to the hospital. The temperature remained at approximately 102°F., and he had profuse night sweats. He noted the onset of hoarseness, which became progressively worse.

Further probing into the past history disclosed that he had had a transient ache in the left elbow and right hip five months prior to admission. His weight had dropped from 177 to 162 lb. in the year prior to admission.

Physical examination showed no significant change from the previous admission, except that he appeared chronically ill and had a temperature of 101°F. The eye condition had improved and was considered well controlled by the Ophthalmology Service.

The blood pressure was 140 systolic, 85 diastolic and showed little change during the hospital course.

Repeated urine examinations revealed specific gravities ranging from 1.005 to 1.015, + to ++ tests for albumin and no sugar. The sediments contained 5 to 10 white cells per high-power field. Early specimens showed no red cells or casts in the sediment, but later ones showed 4 to 10 red cells and occasional cellular casts. The hemoglobin remained at approximately 11.0 gm., and the white-cell count ranged from 7500 to 24,600. The differential on admission revealed 92 per cent neutrophils, 3 per cent lymphocytes, 4 per cent monocytes and 1 per cent myelocytes. The stools were guaiac negative, and stool cultures were negative for pathogens. The albumin was 3.46 gm. and the globulin 1.94 gm. per 100 cc. The nonprotein nitrogen was 29 mg. on the second hospital day and 90 mg. per 100 cc. on the seventh hospital day; it ranged from 70 to 90 mg. on subsequent determinations. The Widal and agglutination tests for brucella and heterophile antigen were reported as negative. Blood cultures were negative.

In the hospital, the patient's temperature remained between 100°F. and 102°F. He complained of migratory joint pains, sore throat and general malaise. Examination of the joints showed no definite changes. On the second hospital day, the throat showed a superficial necrotic lesion about 1 cm. in diameter on the right soft palate, and in-

jected anterior pillars. There were a few small, soft cervical lymph nodes. Roentgenograms of the sinuses showed clouding of the left frontal sinus and antrum, but this was thought to be caused by membrane formation and not by pus. The pharynx, on the following day, showed improvement of the soft-palate lesion. Ophthalmoscopic examination on the third hospital day showed no scleral injection or increased ocular tension. Roentgenograms of the feet and hands, on the fourth hospital day, disclosed good mineralization, thickening of the cortex of each second metatarsal joint, no soft-tissue thickening of the feet and some thickening of the soft tissues about each index finger, in the region of the proximal interphalangeal joint — more marked on the left. A bone-marrow aspiration on the fifth hospital day showed a myelocyte-erythrocyte ratio of 6:1, and an increase in eosinophils. There were no lupus erythematosus cells.

On the tenth hospital day there was an increase in the azotemia and a flare-up of pain, and redness and swelling of the first right metacarpal joint. The sodium was 120.4 milliequiv. per liter, and the eosinophil count was 826 per cubic mm. On the fourteenth hospital day he had a shaking chill, with rise in temperature to 103°F., followed by a drop in temperature to 96°F. and profuse diaphoresis. Four days later he developed a + pitting edema of the ankles. A transient right-facial weakness was noted on the twenty-seventh hospital day, and on the following day a right hemiplegia with right-facial weakness and partial aphasia developed. Later that day he became semicomatose and developed a flaccid quadriplegia. On the following day the comatose state deepened, and he died.

Differential Diagnosis

Dr. Thomas A. Warthin*: If would be helpful if I knew whether he had noted abdominal pain, anorexia or headache at any time during the two weeks prior to the final admission.

Dr. Benjamin Castleman: There was no history of abdominal pain.

Dr. Warthin: Was anything known about the state of the larynx? Was there merely inflammation, or was there evidence of paralysis? If not, I shall assume that it was all inflammatory.

*Assistant in medicine, Massachusetts General Hospital; chief of medicine, West Roxbury Veterans Hospital.

May I see the x-ray films? I hope there are chest films.

Dr. Joseph Hanelin: There are two chest x-ray films, one made four days after the first admission, the second two weeks later. The lung fields appear quite clear. The left ventricle is somewhat large, and the aorta is slightly tortuous. There is obliteration of the left costophrenic sinus, which appears the same on the two films. I think it is due to thickened pleura, rather than to any fluid within the chest.

Dr. Warthin: Does anything about the heart shadows suggest a congenital anomaly of any sort?

Dr. Hanelin: No; it looks more like a hypertensive type of heart.

I do not believe the films of the hands and feet add a great deal. There is just a bit of soft-tissue swelling about the proximal interphalangeal joints of the index fingers, bilaterally. There is a cortical thickening of both metatarsal shafts, and minimal periosteal new-bone formation along the outer aspects of the distal tibial shafts. In the absence of periostitis in the hands and feet, it is unlikely that the changes in the tibias represent pulmonary osteoarthropathy.

Dr. Warthin: No defects in the ribs or chest?

Dr. Hanelin: No.

Dr. Warthin: This fifty-five-year-old man entered the Eye and Ear Infirmary with an eye complaint; that is a little confusing to me. With some aid from the ophthalmologists, I think I can say that undoubtedly he had active sinusitis, and, associated with it or unrelated to it, a low-grade primary glaucoma with a slight but definite increase in tension in both eyes. There was nothing in that degree of tension to cause the pain in the eyes of which he complained. Certainly the episcleral injection is what one would associate with sinusitis, and not with an intrinsic inflammatory disturbance of the eye. Of interest, however, is the fact that the sinusitis was apparently quite severe during that admission. His physicians certainly left no stone unturned as far as chemotherapy and antibiotics went. They also were concerned about the glaucoma, so that he received many miotics and vasoconstrictors. There was no evidence of secondary glaucoma due to a uveal-tract infection. Therefore, I shall discard very quickly any suggestion of sarcoid of the uveoparotid fever type in the background. The x-ray films of the chest and the

physical examination present no evidence of lymphoma or metastatic carcinoma. Brucellosis can be ruled out by the atypical course, and by the negative agglutination test. There was no evidence that he had any unusual fungus infection.

That brings me back to our old friend, the differential diagnosis of conditions of a septic type. There were several congenital or developmental anomalies, so that he may have had a congenital cardiac lesion, with a superimposed bacterial endocarditis. The lack of a cardiac murmur, or evidence of a congenital anomaly upon which an endocarditis might have been superimposed, and the negative blood culture all tend to rule out such a diagnosis.

Miliary tuberculosis is another possibility — which I think we can rule out on the negative x-ray examinations of the chest, since two were taken. One would expect some change in the second examination. Miliary tuberculosis, in my experience, has either a normal or low white-cell count or a myeloid type of reaction with a white-cell count of 40,000 or 50,000. The white-cell counts reported here were high, normal or only moderately elevated. The terminal event of a localized cerebral episode without any previous meningitis is very much against a meningeal infection or intracerebral tuberculoma.

I come, then, to the two best possibilities — namely, the extension of sepsis from the sinuses into the brain, and polyarteritis nodosa. I shall take up the latter first. There are a number of things in favor of polyarteritis. First, in the order of this history, were a fistula, masses of unknown nature that were extracted from the buttock, and recurrent arthralgias. The fact that he was given sulfadiazine during the admission to the Eye and Ear Infirmary points in that direction. During the final hospitalization he developed arthralgias and, apparently, definite lesions of the rheumatoid type in the fingers. He also had azotemia and changes in the urine suggesting that the kidneys had become involved. In addition, the bone marrow showed an increase in eosinophils. However, penicillin and other antibiotics are very likely to do that; and this must be taken into consideration The terminal event certainly sounds like a vascular accident, and is quite a strong factor in favor of polyarteritis. I have, unfortunately, no evidence that a muscle biopsy was performed.

What about local sepsis, with a brain abscess extending from the frontal sinus or the antrum? First, the sinusitis was on the left side and the hemiplegia was on the right — so these went together. Evidently he had a good deal of chemotherapy at the time of the sinusitis, which was stopped when he went home. After a latent period, he came back with sepsis, and finally had focal neurologic signs. That is a typical story for brain abscess. A focus that has not been treated long enough to be eradicated develops, after a latent period, into an obvious local brain lesion. One strong point against it was the short duration of the neurologic signs. They lasted only three days. By and large, a brain abscess does not kill with that degree of rapidity, although with a patient who has been septic for a considerable period of time it is still a possibility. Another point that suggests sepsis is that many patients with *Staphylococcus aureus* infection have peculiar deep-red throats, hoarseness and soft-palate lesions such as are described here; and, since *Staphylococcus aureus* is the organism most likely to be present in a brain abscess, it leads me a little more in that direction. Anyone who has had a septic condition for a long time and has received chemotherapy is entitled to a certain increase in the number of eosinophils in the bone marrow, and I do not think that the increase here should prevent me from making that my primary diagnosis. Therefore, I shall place sepsis with brain abscess over polyarteritis nodosa. I think, if an abscess was present, that it probably was due to *Staphylococcus aureus*. In addition, he should have had a purulent sinusitis, and probably a mild primary glaucoma.

Clinical Diagnosis

Polyarteritis nodosa, with terminal basilar-artery thrombosis.

Dr. Warthin's Diagnoses

Brain abscess, *Staphylococcus aureus*.
?Polyarteritis nodosa.
Sinusitis, purulent.
Glaucoma, primary.

Anatomical Diagnoses

Polyarteritis nodosa.
Cerebral hemorrhage.

Pathological Discussion

Dr. John B. Stanbury: Although the correct diagnosis was suggested by Dr. Christenson at the

time of admission, we tended to neglect that for several days because he had had a chill and because of the strong presumption of sepsis, but that did not pay off. All the blood cultures came back negative, and we obtained what Dr. Warthin was looking for, namely, a muscle biopsy — which helped us immeasurably. The thing that amazed me was the rapid downhill course. After the diagnosis was made, he rapidly fell apart, with swiftly progressing renal impairment and personality change, which was the first indication of the impending cerebrovascular disaster that finally killed him.

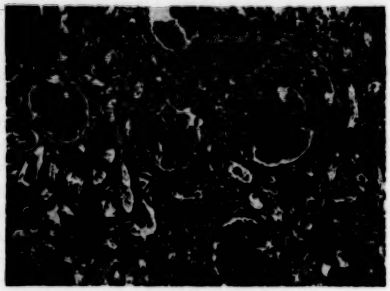

FIGURE 1.

DR. WARTHIN: One thing that led me away from polyarteritis was the fact that he had no increase in blood pressure, no evidence of involvement of the lungs or heart and no gastrointestinal complaints. With polyarteritis, one would expect him to have had at least two of these systems involved. I am interested to see that here was a patient who did not have signs referable to one of these systems.

DR. CASTLEMAN: The muscle biopsy did make the diagnosis of polyarteritis nodosa. There was a marked inflammatory reaction — not only in the walls of the vessels, but throughout the stroma.

At the time of autopsy, the gross findings were practically negative, except for the kidneys. The heart weighed about 400 gm., and did not seem to be hypertrophied. The kidneys weighed 700 gm. — about twice the normal size. The surface of the kidneys was peppered with petechial hemorrhages. These were present throughout the cortex and medulla. It was surprising that a man with polyarteritis nodosa, and with kidneys looking like this, should have a nonprotein nitrogen of only 70 to 90 mg. Microscopically, there was marked cellular and fibrinoid necrosis of some of the tufts (Fig. 1). The cellular infiltration extended throughout the stroma. In some glomeruli, in addition to the fibrinoid necrosis, crescent formation was beginning. This appearance in the kidneys, which at first glance would make you think of a chronic or subacute glomerulonephritis, has been described in polyarteritis nodosa — especially in the cases with no hypertension. It is remarkable that there were very few arterial lesions in the kidneys, in contrast to the other organs, which had many. Davson and his co-workers* reported a series of patients with periarteritis nodosa in which they divided the renal conditions into two groups: those who had a picture similar to the one under discussion — fibrinoid necrosis of the tufts, nephritis of the embolic type (because the lesions are focal) and few if any arterial lesions, absence of the usual clinical picture of nephritis, and without hypertension; and those with many arterial lesions, hypertension and focal areas of infarction as a result of the arterial thrombosis. This case apparently falls into the first group. The terminal incident was a cerebral hemorrhage.

*Davson, J., Ball, J., and Platt, R. Kidney in periarteritis nodosa. *Quart. J. Med.* 17:175-202, 1948.

The New England
Journal of Medicine

Formerly
The Boston Medical and Surgical Journal
Established 1828

Official Organ of
THE MASSACHUSETTS MEDICAL SOCIETY
and
THE NEW HAMPSHIRE MEDICAL SOCIETY
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MICROFILM volumes available to regular subscribers through University Microfilms, 313 N. First Street, Ann Arbor, Michigan.

MATERIAL, other than original articles, should be received not later than noon on Thursday, three weeks before date of publication.

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COMMUNICATIONS should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts. Telephone KE 6-2094.

ANOTHER MEDICAL SCHOOL?

IN HIS second Inaugural Address, delivered on January 4, Governor Paul A. Dever recommended the establishment of medical and dental schools under the auspices of the University of Massachusetts, to be located in the City of Boston where, he said, he had been assured "a competent and adequate staff is available." In so far as the recommendation concerns a medical school, the profession is naturally interested in discussing it.

One of the Governor's statements was that there is "an acute and growing shortage of physicians . . . within the Commonwealth." No facts are given in support of this. As a general statement this is similar to those sweeping pronouncements that have been made so frequently that they are now part and parcel of the trite phraseology of socialized medicine. The truth is that the number of doctors graduating from approved medical schools in the United States has been for thirty years increasing more rapidly than the population. This truth has been patiently stated on many occasions. Looking back over the numbers of annual graduates, one finds that they have more than doubled since 1920; the actual figures for each fifth year may be tabulated as follows to show the nationwide production of physicians.

1920	2,680
1925	3,842
1930	4,565
1935	5,101
1940	5,097
1945	5,136
1950	5,553

It is true that doctors now function in more diverse ways than formerly, but in view of these figures it is difficult to be sure of an over-all shortage. A superabundance of physicians also indicates hazards for the public, especially if quantity is achieved at the expense of quality, but there can be no doubt that so far, in this Commonwealth, their quality has improved fully as much as their quantity during this thirty-year period. In wartime, of course, there will always be a shortage of physicians, as there are shortages of everything.

When one turns from the countrywide supply of physicians to those now available to the citizens of Massachusetts, it is found that there is only one state in the Union with more doctors per population unit than Massachusetts. According to the figures in the 1950 directory of the American Medical Association, there are in Massachusetts 538 people for each physician. The rest of the New England states have the following population for each physician: New Hampshire, 733; Maine, 969; Connecticut, 612; Rhode Island, 758; Vermont, 648. New York alone has fewer people per physician, the number being 475. As we go farther afield we find Pennsylvania with 740, Ohio with 808, Michigan with 914, Illinois with 660, Virginia with 648 and California with 639. Oklahoma, Tennessee and North Dakota all have more than 1000, while Texas has slightly fewer. Of course, these

figures indicate the ratios for the total numbers of physicians, some of whom are naturally not in practice for various reasons, but the comparison of one state with another would seem to be valid, especially when the variation is a wide one. When one reflects upon the fact that Massachusetts, with no tax subsidization of medical education, compares in this way with the other states mentioned, nine of which maintain substantial tax subsidization of medical education, one cannot refrain from questioning the wisdom of asking the Massachusetts taxpayers to embark upon what would most assuredly be an expensive and difficult enterprise.

Other questions come to mind, as they undoubtedly have come to the mind of the Governor — for he stated that medical education can never be cheap and that he knows that the "details required to be solved . . . are manifold." The *Journal* will gladly open its columns to a discussion of Mr. Dever's recommendation, for it deserves the widest possible application of professional thought — economic and legal as well as medical. In the outcome, may common sense prevail!

AMERICAN MEDICAL EDUCATION FOUNDATION

THOSE persons to whom the destinies of the American Medical Association are entrusted have been planning with considerable concern the next step to be taken in the program for medical education that was inaugurated by the trustees in December.

The appropriation of a half million dollars for this purpose that was made at that time was no idle gesture, nor was it taken as such by the majority of thoughtful physicians. Almost immediately the fund was augmented by gifts from many sources, of particular significance being the contributions voluntarily made by a number of practicing physicians, of whom the members of the American Medical Association are the most representative.

That next step, which demonstrates to all open minds the sincerity of the Association, consists, as announced on January 20 in the *Journal of the American Medical Association*, of the establishment of the American Medical Education Foundation as a nonprofit corporation under the laws of Illinois. Its purpose is to receive the continuing contributions to the fund, already so auspiciously started, that will come from individual members and friends of the medical profession, and to make distributions from it.

The directors of the fund are representatives of the board of trustees, the officers and the Council on Medical Education and Hospitals of the American Medical Association. The use of funds, once they are allocated to approved medical schools, will be unrestricted.

The Foundation will co-operate with any other major efforts that may be made to raise money from voluntary sources, reserving for its own special efforts the practicing physicians of the country, most of whom may be considered still to owe some debt to society for their education. In the discharge of this debt an annual contribution of $100 from each physician is considered not unreasonable, as was, in fact, suggested by Currens in his letter published in the *Journal* on December 28, 1950.

The sum of $100 is not, of course, taken as the *sine qua non* of indebtedness; smaller sums will be acceptable, but it is expected that no physician will try to placate his conscience in the matter with a token contribution.

All physicians have now their opportunity of helping to decide the future of medical education. Can it continue as a free enterprise, or must it tread the path that leads to eventual government control, as is so ardently desired by the proponents of the welfare state?

THE CONCORD PLAN

WHEN an atom bomb exploded over Hiroshima on August 6, 1945, and in a single moment an entire city fell in ruins, a pall of fear settled over the world. This fear has acquired a singularly hopeless quality because of the repeated assertion that there can be no defense against the weapon. It has been fostered by premature and inaccurate reports of bizarre blood dyscrasias, mass sterility and altered germ plasm to be wrought in entire nations by the uncontrollable radioactivity produced by the bomb. This background of fear has made

it difficult to face the actuality of an atomic war.

Fortunately, there are many rays of encouragement and light in this dark hour. It is now recognized that even an atom bomb has its limitations. It has little or no penetration, so that beyond the area of intense heat shelters are amazingly effective. The radioactive effects of an air burst are spent in a matter of seconds. An underwater burst will play havoc with port installations, but it will do little harm to the civilian population of harbor cities. Moreover, the bomb must be delivered to be effective; and, as Vannevar Bush[1] has pointed out, the mass air raids of the last war will be suicidal against a prepared defense. Single bombers may get through, but the destruction en route of a plane carrying an atom bomb would be a grievous blow to the economy of even an enslaved nation. The importance and effectiveness of an organized medical defense also have been stressed recently in the *Journal*,[2] and elsewhere in this issue is presented what may be regarded as the most effective answer to an atomic attack: namely, an organized community defense.

The details of the "Concord Plan" are not important. If necessary, these can be modified to meet state or federal recommendations as more information becomes available. The significant fact is that Concord as a community has taken upon itself the responsibility for its own care, and for the care of neighboring areas that might be so unfortunate as to be hit. The people of Concord have manifested their determination to resist. They have not waited for outside help, and they are not expecting to lean on others or to flee to urban areas for assistance. Every city, every town, every hospital, every factory, every business, every school and every home must do likewise.

An elementary knowledge of first aid, simple shock therapy and fire-fighting can be acquired by every citizen. Every neighborhood and home must have a plan for living for several days without the usual sources of food, water, heat and light. Every street must have its organized fire fighters and first-aid teams, but in addition no other citizen can shun the responsibility for acquiring a basic knowledge of these measures. To have something to do, however simple, and to know how to do it, are the antidotes for panic. All planning must be integrated with state and federal efforts, but the responsibility is individual. Like the citizens of Concord, everyone must recognize his duty, accept the challenge and calmly await the test.

It is of historic significance that Concord has taken this action. For this spirit, this will for self-preservation and self-help, is the essence of civil defense. Just as once before in our history such determination found its practical outlet in Concord and spread throughout the nation, so it will again if the need arises.

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1. Bush, V. *Modern Arms and Free Men.* 273 pp. New York: Simon & Schuster, 1949.
2. Currier, D. E., and Bradford, C. H. Presentation of basic defense program. *New Eng. J. Med.* 244:75-81, 1951.

AN APPEAL TO THE HEART

The American Heart Association, in making its annual February campaign for funds, has published a list of Ten Commandments for cardiac patients. The first of these in particular will appeal to the professional heart: "Find a doctor you respect, obey him, and cling to him as though he were your most precious possession, as he is."

Other commandments are equally pertinent, although the first, if faithfully observed, covers the nine: "Pity the fat man; the statistician numbers his days," "When the snow drifts high, let some younger fellow shovel out the driveway," "He who doctors himself has a fool for a physician" and, finally, "Be your age."

So the effort to lengthen man's days continues — laudably, if the lengthening can be accompanied by comfort, a degree of security, some sense of continued usefulness, and not too great a lengthening of the shadows, also. If one is to be encouraged to live, then he should at least receive some protection from the chill wind that so often rises as the sun goes down.

The national financial goal to be achieved this month is $6,000,000; as part of this, to carry on its own work, the Massachusetts Heart Association and its twelve affiliated chapters are asking for $350,000. The sums to be raised in the six New England states aggregate approximately a quarter of a million dollars; and in this connection it is to be remembered that when the New England Heart

Association was separated last year into the six component state associations, the membership refused to abandon its regional loyalty, continuing a corporate existence as the New England Cardiovascular Society.

A large part of the money raised will be used for the support of research, other funds being allocated to the study of services, to supply penicillin to patients with subacute bacterial endocarditis, to publish and distribute the "Cookbook for Low-Sodium Diet," and to carry on various activities in education and rehabilitation.

There is a difference between great-heartedness and cardiac enlargement. The former, arising from the spirit, can often be of assistance to the latter, struggling against its physical limitations. As David implored, "Let my heart be sound in thy statutes; that I be not ashamed."

A philosophical invention from Whitby appears in the form of a Tempest Prognosticator, whose accuracy is said to have been tested by the storms of the last twelve months. The inventor is Dr. Merryweather. (Illustrated London News.)

Boston M. & S. J., *February 5, 1851*

MASSACHUSETTS MEDICAL SOCIETY

CHANGE IN THE RESTRICTIONS ON THE REFILL OF PRESCRIPTIONS

Additional Report from the Committee on Pharmaceutical Problems

On December 5, 1950, Mr. Oscar R. Ewing, federal security administrator, proposed a new regulation intended to eliminate misunderstandings about the restrictions on the refill of prescriptions.

Mr. Ewing's statement implied that the new regulation would relax the restriction on the refill of so-called over-the-counter drugs — those that may be freely sold without prescriptions. There will be no relaxation in regard to "prescription drugs," including those classified as dangerous drugs (for example, barbiturates, sulfa drugs and thyroid tablets), and preparations labeled by the manufacturer as to be dispensed only on a physician's prescription. "We will continue to press legal action," said Mr. Ewing, "against those few pharmacists who endanger their customers' lives by selling these toxic drugs without prescription or upon unauthorized refill."

Physicians are, therefore, again reminded that they should give complete directions when prescribing any of these "prescription drugs." If the prescription is not to be refilled, it should be marked N.R. If the physician intends that the patient receive renewed supplies, he should mark the prescription "To be refilled as needed _____ times" or "To be refilled as needed for _____ months." Under the present regulations, a prescription without such a notation is presumed to be nonrefillable.

Prescriptions written in accordance with these recommendations should cause no difficulties. However, in the matter of prescriptions previously issued without such precise directions, physicians should co-operate willingly with pharmacists to ensure proper treatment of their patients. Pharmacists report that they are too frequently called upon to explain what the physician intended, or that they find themselves in the position of defending the physician's action in regard to a prescription. The pharmacist should be relieved of this unwelcome responsibility. The physician should prevent resentment or misunderstanding on the part of the patient by explaining the nature of the prescription when he writes it. He should tell the patient what is to be expected in regard to future treatment and in regard to the refill of the prescription, if necessary, in the future.

A patient referred to his physician for renewal of a prescription without knowing in advance that this might be needed has sometimes thought that there has been an attempt to make money out of his case by collusion of pharmacist and physician. This suspicion has been strengthened when a fee has been charged by the doctor. Patients may actually be at fault in failing to state that the purpose of a call is merely to obtain authorization for refill of an old prescription.

It is therefore recommended by the committee that a physician asked by a druggist or a patient for authorization of the refill of a prescription should make the authorization without charge when this is the only service involved.

The new regulation proposed by Mr. Ewing has been challenged by pharmacists. New legislation is still before Congress. Meanwhile, physicians are reminded to deal carefully with prescriptions, in conformity with the government's present interpretation of existing laws.

Charles L. Brennan, M.D.
George L. Maison, M.D.
Frank N. Allan, M.D., *Chairman*

CIVIL DEFENSE ORGANIZATION

The following material, released on Civil Defense Bulletin No. 1, has been submitted for the information and action of district medical society presidents, secretaries, and emergency medical service committee members by Leland S. McKittrick, M.D., President, Massachusetts Medical Society, and Donald Currier, M.D., Chairman, Committee on Emergency Medical Service, Massachusetts Medical Society:

At the state level the civil defense organization is made up of 7 divisions, each of which is headed by a chief. These are as follows: Medical and Health — Vlado A. Getting, M.D.; Rescue, Transportation, and Evacuation; Police; Fire; Communications; Utilities; and Civilian War Aid.

The Medical and Health Division is subdivided into 12 sections, each of which is headed by a chief, as follows: First Aid — Donald Currier, M.D.; Hospital and Medical — Charles G. Hayden, M.D.; Blood — Lamar Soutter, M.D.; Laboratory —Geoffrey Edsall, M.D.; Vital Statistics — Arthur Hassett, Secretary of the Commonwealth; Mortuary — Richard Ford, M.D.; Communicable Disease Control — Conrad Wesselhoeft, M.D.; Monitor — Worthen Taylor, Department of Sanitary Engineering; Biological and Chemical Defense — John C. Snyder, M.D.; Environmental Sanitation — Clarence Sterling, Department of Public Health; Food and Nutrition — Fredrick Stare, M.D.; Health and Supplies — Louis Adams.

Each of the sections of the Health and Medical Division is sponsored by several voluntary and governmental agencies. The Massachusetts Medical Society is one of the sponsors of the following sections: First Aid and Ambulance, Hospital and Medical, Blood, Vital Statistics, Communicable Disease Control.

The Hospital and Medical Section, which will head up planning and operations for most physicians, dentists and auxiliary personnel, is sponsored by the following agencies, each of which has designated a representative as indicated: Massachusetts Medical Society, Harold M. Frost, M.D.; Massachusetts Hospital Association, Henry G. Brickman (temporary); Massachusetts Dental Society, Arno Bommer, D.M.D.; Massachusetts Osteopathic Society, H. Earle Beasley, D.O.; Massachusetts State Nursing Association, Rita Kelleher, R.N.; Licensed Attendants Association of Massachusetts, Mrs. Lula A. Snow; Massachusetts Physical Therapist Association, Miss Joan McBratney; Department of Public Welfare, Miss Virginia Vahey; Department of Correction, Samuel A. Merlin, M.D.; Department of Mental Health, Malcolm Farrell, M.D.; Division of Registration in Medicine, Bancroft Wheeler, M.D.; Division of Registration in Dentistry, Raymond J. Nagle, D.M.D.; Division of Registration in Nursing, Miss Eleanor Bowen; American National Red Cross, William B. Freeman, M.D.

The designated representatives of the several agencies sponsoring the Hospital and Medical Section will constitute an executive committee for the section. In addition, the chief of the section, Dr. Hayden, has asked that advisory committees be appointed or designated by the sponsoring agencies that deal directly with personnel who will participate in the event of an emergency, namely: Massachusetts Medical Society, Massachusetts Hospital Association, Massachusetts Dental Society, Massachusetts Osteopathic Society, Massachusetts State Nursing Association, Licensed Attendants Association of Massachusetts, American National Red Cross, Massachusetts Physical Therapist Association.

The Chief of the Hospital and Medical Section has also asked the members of the Blue Shield Fee Committee to serve as consultants on mass treatment of casualties.

For civil defense purposes the State of Massachusetts has been divided into 9 civil defense regions. Each region will be organized to correspond, more or less, with the state level organizational pattern. The directors of the 9 regions are as follows: Mayor Robert Capeless of Pittsfield, Region I; Mayor Daniel B. Brunton of Springfield, Region II; City Manager Everett F. Merrill of Worcester, Region III; City Manager John J. Flannery of Lowell, Region IV; Mayor John B. Hynes of Boston, Region V; Mayor Melvin B. Clifford of Brockton, Region VI; Chairman of the Board of Selectmen of Barnstable, Victor F. Adams, Region VII; Mayor William P. Grant of Fall River, Region VIII; Mayor Peter J. Levanti of Fitchburg, Region IX.

Co-ordination of medical and health activities in the 9 civil defense regions will be under the general direction of a trained health officer attached to the State Department of Public Health: Region I, Charles E. Gill, M.D., 184 North Street, Pittsfield; Region II, Walter W. Lee, M.D., Marshall Hall Annex, Amherst; Region III, Kenneth I. E. MacLeod, M.D., 476 Main Street, Worcester; Region IV, Walter J. Pennell, M.D., 367 Main Street, Wakefield; Region V, Leon Sternfeld, M.D., 227 Commonwealth Avenue, Boston; Region VI, Grace Lutman, M.D., 1245 Hancock Street, Quincy; Region VII, Fred L. Moore, M.D., Health Officer, Barnstable County Health Department, Court House, Barnstable; Region VIII, Harold W. Stevens, M.D., 105 William Street, New Bedford; Region IX, Arthur E. Burke, M.D., 17 Putnam Street, Fitchburg.

The 9 civil defense regions are not co-terminous either with individual district medical society boundaries or combinations of such boundaries. The following table shows the relation between the

9 Civil Defense Regions and the 18 medical society districts.

REGION I

BERKSHIRE

REGION II

FRANKLIN, except for	
Warwick	Region IX
Orange	IX
Wendell	IX
New Salem	IX
HAMPSHIRE, except for	
Plainfield	Region I
Cummington	I
Middlefield	I
HAMPDEN, except for	
Chester	Region I

REGION III

WORCESTER, except for	
Hardwick	Region II
Warren	II
Hubbardston	IX
Harvard	IX

REGION IV

ESSEX NORTH

ESSEX SOUTH

MIDDLESEX NORTH, except for	
Pepperell	Region IX
Groton	IX
Littleton	IX
Boxborough	IX
Acton	IX
Bedford	V

REGION V

MIDDLESEX EAST, except for	
Burlington	Region IV
Wilmington	IV
North Reading	IV
MIDDLESEX SOUTH, except for	
Stow	Region IV
Maynard	IV
Concord	IV
Hudson	III
Sudbury	III
Wayland	III
Marlborough	III
Framingham	III
Natick	III
Ashland	III
Hopkinton	III
Holliston	III
Sherborn	III

SUFFOLK

NORFOLK, except for	
Medway	Region III
Billingham	III
Plainville	VIII
Millis	VI
Medfield	VI
Franklin	VI
Norfolk	VI
Wrentham	VI
Foxborough	VI
Sharon	VI
Walpole	VI

NORFOLK SOUTH

REGION VI

PLYMOUTH, except for	
Scituate	Region V

REGION VII

BARNSTABLE

REGION VIII

BRISTOL NORTH, except for	
Mansfield	Region VI
BRISTOL SOUTH, except for	
Martha's Vineyard	Region VII
Nantucket	VII

REGION IX

WORCESTER NORTH

DEATHS

DUMPHY — John J. Dumphy, M.D., of Worcester, died on December 19. He was in his fifty-fifth year.

Dr. Dumphy received his degree from Harvard Medical School in 1921. He was formerly chairman of the Worcester Board of Health and president of Worcester District Medical Society and was a member of the staffs of St. Vincent, Belmont, Harrington Memorial, Milford and Holden District hospitals and a fellow of the American College of Physicians and the American Medical Association.

His widow survives.

FRAIM — Irving W. Fraim, M.D., of Waltham, died in Brunswick, Georgia, on January 18. He was in his sixty-second year.

Dr. Fraim received his degree from Tufts College Medical School in 1913. He was a fellow of the American Medical Association.

His widow, a sister and two brothers survive.

POWELL — Charles A. Powell, M.D., of Boston, died on December 28. He was in his sixty-fourth year.

Dr. Powell received his degree from Boston University School of Medicine in 1913. He was formerly assistant instructor in tropical diseases at Boston University School of Medicine and assistant administrator and staff member of the Massachusetts Memorial Hospitals and was a fellow of the American Medical Association.

His widow, two daughters and seven grandchildren survive.

ROBBINS — William B. Robbins, M.D., of Boston, died on January 16. He was in his seventy-fourth year.

Dr. Robbins received his degree from Harvard Medical School in 1904. He was a fellow of the American Medical Association.

His widow, three daughters, three sons and his twin brother survive.

SCOBORIA — Arthur G. Scoboria, M.D., of Chelmsford, died on December 28. He was in his seventy-ninth year.

Dr. Scoboria received his degree from Tufts College Medical School in 1895 and from Harvard Medical School in 1898.

His widow and two daughters survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

MASSACHUSETTS PREMARITAL-EXAMINATION LAW AND INTERSTATE MARRIAGES

NICHOLAS J. FIUMARA, M.D., M.P.H.*

The recently amended premarital-examination law† in Massachusetts has made it possible to simplify some of the problems of United States and Canadian residents who plan to be married in Mas-

*Director, Division of Venereal Diseases, Massachusetts Department of Public Health.

†Fiumara, N. J. Premarital-examination law. *New Eng. J. Med.* 243: 238-240, 1950.

sachusetts. Hitherto, the law has recognized only the Massachusetts premarital certificate, a restriction that has often proved exceedingly awkward for out-of-state residents. This certificate could be signed by a physician licensed in the United States only after the patient's blood test for syphilis was performed at a state or federal public-health laboratory or a laboratory of the armed forces. The examination reports of Canadian physicians and the provincial laboratories were not acceptable.

The new law will permit the city and town clerks in Massachusetts to accept certificates from 34 states, New York City, 2 territories and 1 Canadian province, provided these certificates are signed by a physician licensed to practice in the areas mentioned and provided, also, that the blood test for syphilis

Table 1. *Reciprocity Between Massachusetts and Individual States, Territories, New York City, and Canadian Provinces.*

State, Territory or Province	Massachusetts Certificate Acceptable	Out-of-State Certificate Acceptable in Massachusetts	Waiting Period in Days Between Application for and Issuance of License	No. of Days Blood Test Valid in Listed State
United States:				
Alabama	No	Yes	0	30
Alaska	Yes	Yes	3	30
Arizona	No law		0	
Arkansas	No law		0	
California	Yes	Yes	3	30
Colorado	Yes	Yes	0	30
Connecticut	Yes	Yes	5	35
Delaware	Yes	Yes	1 residents 4 nonresidents	30
District of Columbia	No law		5	
Florida	No	Yes	3	30
Georgia	No	Yes	0	30
Hawaii	No	Yes	3	30
Idaho	Yes	Yes	0	30
Illinois	No	Yes	1	15
Indiana	No	Yes	0	30
Iowa	Yes	Yes	0	30
Kansas	Yes	Yes	3	30 residents 20 nonresidents
Kentucky	No	Yes	3	15
Louisiana	Yes	No*	3	15
Maine	Yes	Yes	5	30
Maryland	No law		2	
Michigan	No	Yes	5	30
Minnesota	No law		5	
Mississippi	No law		0	
Missouri	Yes†	Yes	3	15
Montana	Yes	No	0	20
Nebraska	No	Yes	0	30
Nevada	No law		0	
New Hampshire	Yes	Yes	5	30
New Jersey	Yes	Yes	3	30
New Mexico	No law		0	
New York State	Yes	Yes	0	30
New York City	Yes	Yes	3	30
North Carolina	No	Yes	3	30
North Dakota	No	Yes	0	30
Ohio	No	Yes	5	30
Oklahoma	No	Yes	0	30
Oregon	No	Yes	3	10
Pennsylvania	No	Yes	3	30
Puerto Rico	No	No		10
Rhode Island	Yes	Yes	0 residents and male nonresidents 5 nonresident females	40
South Carolina	No law		1	
South Dakota	No	Yes	0	20
Tennessee	No	Yes	3	30
Texas	No	Yes	0	15
Utah	No	Yes	0	30
Vermont	Yes	Yes	5	30
Virginia	No	No	0	30
Virgin Islands	No law			
Washington	No law		3	
West Virginia	No	Yes	3	30
Wisconsin	No	No	5	15
Wyoming	No	Yes	0	30
Canada:				
Alberta	No	No		14
British Columbia	Law never put into effect			
Manitoba	Yes	No	0‡	30
New Brunswick	No law			
Newfoundland	No law			
Nova Scotia	No law			
Ontario	No law			
Prince Edward Island	No	No	7	30
Quebec	No law			
Saskatchewan	No	Yes		30

*No official certificate prescribed.

†Attach Wassermann Laboratory slip to Massachusetts Form.

‡Special authorization required for out-of-state residents. Out-of-state premarital examination certificates acceptable in Massachusetts must be signed by a physician licensed to practice in any of the states, the District of Columbia or territories of the United States or a medical officer of the armed forces or Public Health Service. The blood test for syphilis must have been performed at a laboratory operated by, or for, a state department of public health, District of Columbia, New York City or territorial health department of the United States, laboratories of the armed forces and Public Health Service of the United States, or the laboratories of the provincial health departments of Canada.

is performed in the laboratory of a state, New York City, or territorial health department of the United States or provincial health department of Canada.

Military personnel may be examined by a medical officer of the armed forces, and their blood samples tested at an armed-forces laboratory. Also acceptable are the examinations of a medical officer of the Public Health Service and the reports of its laboratories. In either case, the Massachusetts certificate is to be completed for intrastate military personnel and those stationed in the states and territory that have no premarital law or whose certificates are not yet acceptable in Massachusetts. The certificates of the states and territories acceptable in Massachusetts may be used by a medical officer of the armed forces or Public Health Service.

TABLE 2. *List of States, Territories and Canadian Provinces Requiring Own Premarital Certificate but Accepting the Laboratory Report of State Wassermann Laboratory and Physical Examination of Massachusetts Physicians (December 1, 1950).*

STATE, TERRITORY OR PROVINCES	MASSACHUSETTS STATE WASSERMANN LABORATORY REPORT ACCEPTABLE	MASSACHUSETTS PHYSICIAN'S PHYSICAL EXAMINATION REPORT ACCEPTABLE
United States:		
Alabama	Yes	Yes
Florida	Yes	Yes
Georgia	Yes	Yes
Hawaii	Yes	Yes
Illinois	Yes	Yes
Indiana	Yes	Yes
Kentucky	Yes	No
Michigan	Yes	Yes
Nebraska	Yes	Yes
North Carolina	Yes	No
North Dakota	Yes	Yes
Ohio	Yes	No
Oklahoma	No	No
Oregon	Yes	No
Pennsylvania	Yes	No
Puerto Rico	Blood test not required	No
South Dakota	Yes	Yes
Tennessee	Yes	Yes
Texas	Yes	Yes
Utah	Yes	Yes
Virginia	Yes	Yes
West Virginia	Yes	No
Wisconsin	Yes	Yes
Wyoming	Yes	No
Canada:		
Alberta	No	No
Prince Edward Island	No	No
Saskatchewan	Yes	No

For all persons, however, both intrastate and out-of-state residents, planning to be married in Massachusetts, the physical examination and blood test for syphilis is valid for thirty days prior to the issuance of the license.

Table 1 demonstrates that as of December 1, 1950, there were 39 states and 3 territories with premarital-examination laws. Massachusetts will accept the medical certificates, when properly executed, of 34 states, 2 territories and New York City. There are 4 out of 10 Canadian provinces that also have such laws in force. At present only the certificate of 1 Canadian province is acceptable in Massachusetts.

In turn, 16 out of 39 states, 1 territory, New York City and 1 Canadian province have agreed to accept the Massachusetts certificate signed by a Massachusetts physician, provided the blood specimen is examined at the State Wassermann Laboratory. It is expected that in time many more states, territories and Canadian provinces will accept the Massachusetts certificate.

Fourteen states and Hawaii, although not accepting the completed Massachusetts certificates, will accept the findings of the Massachusetts physicians and the State Wassermann Laboratory provided that their own forms are used (Table 2).

SUMMARY

As of December 1, 1950, Massachusetts has agreed to honor the certificate forms of 34 states, 2 territories and 1 Canadian province. In return, 16 out of the 39 states and the Territory of Hawaii have agreed to honor the Massachusetts Premarital Examination Certificate form when properly completed.

CORRESPONDENCE

ADDITIONAL CASE OF RICKETTSIALPOX IN BOSTON

To the Editor: Regarding the paper by Pike et al. entitled "Rickettsialpox: Report of a serologically proved case occurring in a resident of Boston," which appeared in the December 7 issue of the *Journal*, I should like to say that I saw a case of rickettsialpox in April, 1947. The diagnosis at that time was made by Dr. Louis Weinstein, of the Haynes Memorial Hospital.

SAMUEL KAGAN, M.D.

Boston

DIABETES DETECTION IN WELLESLEY

To the Editor: With the change of emphasis in public-health work from the prevention of communicable diseases to the detection of chronic illnesses that may be cured or controlled, there is a growing need for closer understanding and more team play between local boards of health and practicing physicians. The time, fortunately, has in general passed when physicians believe that public-health work encroaches upon their prerogatives. They have come to appreciate that the educational work that has been done — for example, in diphtheria-immunization programs or x-ray case-finding projects in tuberculosis work — has brought patients to their offices. Public-health officers have no intention and no desire to practice medicine, but in this era of preventive medicine are solely concerned with getting people immunized and persuading those who need medical care to seek it.

National Diabetes Detection Week presented an excellent opportunity for the combined talents and resources of organized public health and private medicine to join forces to make this project a success. The public-health organizations know — or should know — how to arouse public interest through general publicity and health-education programs, and they can set up the machinery, in co-operation with the medical profession, for mass screening tests. Once this has been accomplished the practicing physician takes over. The health department refers all persons who show glycosuria to their own doctors, or helps them find a doctor if they are newcomers in a town. The public-health nurse may help in follow-up efforts to get hesitant or procrastinating patients to the doctor's office.

Case finding is a rewarding effort in diabetes because of the number of unrecognized cases that exist and the certainty of the benefits of control measures when patients are under

adequate medical supervision. I am writing this message chiefly because, as nearly as I can learn, the opportunities presented by this splendid project this fall were not taken full advantage of, at least in Greater Boston. Furthermore, the publicity given on the national and state level was woefully lacking. Medical journals scarcely mentioned Diabetes Week and the Boston daily papers gave scant attention to it, mentioning chiefly the Diabetes Fair only. The little material that was made available in the form of posters or leaflets was wholly inadequate.

A very modest venture undertaken in Wellesley, a town of 21,000 population, may be of some interest in suggesting what might be done another year if local health departments were urged to co-operate by the medical profession.

About four weeks before the week of November 12 to 18 the local board of health endorsed the idea of co-operating in Diabetes Detection Week. To give the project further citizen backing the Health Division of the Community Council was asked for its endorsement, and a steering committee of physicians and laymen was appointed. All local physicians were informed of the plans and were given a chance to object, or to offer suggestions. Subsequently, the doctors were asked to examine without charge urine specimens that were brought to their offices during the Detection Week, and we later learned that the Norfolk County Medical Society had voted to do this. In addition the Board of Health planned to test specimens, and gained the promise of the ten drugstores in town to distribute and receive specimen bottles, together with instructions, which we furnished. This provided almost ideal decentralized stations, convenient for all parts of the town. It should be added that there was no hospital or outpatient clinic in Wellesley that could be used for testing.

We employed every possible medium of publicity. The local paper was most generous in running several front-page articles, which we furnished, an interview, an editorial and spot announcements. A paid display advertisement was contributed by certain private agencies. Two local radio stations made spot announcements and gave time for a physician member of the committee to broadcast. The posters, fliers and other exhibit material that we could obtain were displayed throughout the town in stores and other places.

We used the Galatest for the Board of Health examinations. Specimens, properly labeled, in the containers we had furnished, were collected twice daily from each pharmacy. The first day's results were a little discouraging, but the number of samples collected pyramided until the last day, when nearly 200 were tested. The Board of Health laboratory tested a total of 393 specimens. The positive findings, which we emphasize were screening tests only so far as the health laboratory was concerned, were as follows:

Result	Male Subjects	Female Subjects
Trace	13	18
+	8	6
++	4	1
+++	3	2
++++	2	3
Totals	30	30

The physicians named by these 60 persons who showed a trace or more of sugar in their urine were immediately notified and were requested to inform us of the results of their final diagnosis on a form that we furnished. To date we have received reports on 24 patients, 3 of whom have diabetes. Of these 2 were new cases, and 1 was a patient with previously known diabetes whose treatment had lapsed.

The physicians have reported 99 specimens left at their offices and examined free. Eight of these disclosed glycosuria. We did not ask that they report their diagnoses so we shall never have that information. One case of diabetes previously known was, however, reported.

The final score, then, was as follows: 482 specimens examined, 38 showing glycosuria; 2 new cases of diabetes; and 2 old cases brought under supervision. There are still 36 patients referred not yet reported upon among whom it seems reasonable to expect that 1 or more cases of diabetes will be found.

The co-operation of physicians, business people and the general public in this project was excellent. It required much extra time and effort on the part of the personnel of a small local department, but in terms of cash expended it cost only $41.40. The results are most rewarding, since at least 2 persons who did not know that they had diabetes are now under medical care. There may be others who have sought, or will seek, diagnosis as the result of our publicity of whom we shall never know.

I should like to urge that next year the physicians responsible for this most worthwhile intensive week of effort to discover unknown diabetes keep in mind the aid they may receive from local boards of health. This, incidentally, is only one area of common interest in which the private doctor and official health agency may co-operate to mutual advantage.

Curtis M. Hilliard, *Supervisor*
Board of Health
Wellesley, Massachusetts

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

History Taking. By George Blumer, M.D. 12°, paper, 51 pp. New Haven, Connecticut: Associates of the Yale Medical Library, 1949. $1.50.

Dr. Blumer was formerly David P. Smith Clinical Professor of Medicine (1920–1940) and dean of the Medical School (1910–1920) of Yale University, and is now professor of medicine, emeritus, and chairman of the Associates of the Yale Medical Library. This small pamphlet, reprinted from the *Connecticut State Medical Journal* as a tribute to Dr. Blumer by his associates and friends, is printed in de luxe style.

Saw-ge-mah (*Medicine Man*). By Louis J. Gariepy, M.D. 12°, cloth, 326 pp. [St. Paul, Minnesota:] Northland Press, 1950. $3.00.

This novel portrays the life of a young country doctor who surmounted all obstacles and went on to success.

NOTICES

ANNOUNCEMENT

Dr. Richard P. Stetson announces the removal of his office to 203 Commonwealth Avenue, Boston.

HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society will be held in the amphitheater of the Peter Bent Brigham Hospital on Tuesday, February 13, at 8 p.m.

The program, presented by the departments of Pathology and Pediatrics, Harvard Medical School and Children's Medical Center (Dr. Charles A. Janeway presiding), will be as follows:

Lipoprotein in Gaucher's Disease, Dr. L. L. Uzman.
Sodium Deficit Produced by Uretero-arachnoid Anastomosis. Drs. W. M. Wallace, D. D. Matson, R. Schwartz and W. H. Bergstrom.
Control of Effects of Antigen-Antibody Reactions on the Tissues. Drs. R. J. P. Wedgwood and C. A. Janeway.

BETA KAPPA CHAPTER OF NU SIGMA NU

The annual lecture of the Beta Kappa (Harvard) Chapter of Nu Sigma Nu Fraternity will be given on Wednesday, February 14, at 5:15 p.m., Amphitheater E, Harvard Medical School. Dr. C. P. Rhoads, of the Sloan-Kettering Institute for Cancer Research and the Memorial Hospital, New York City, will speak on "Progress in Cancer Control." All those interested are cordially invited to attend.

SOUTH END MEDICAL CLUB

A luncheon-meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, February 20, at 12 noon.

Drs. Dwight Emary Harken, thoracic surgeon, Peter Bent Brigham Hospital, and James F. Dickson will speak on the subject "Heart Surgery — A Moving Picture Demonstration."

Physicians are cordially invited to attend.

SPRINGFIELD HOSPITAL

The educational program of the Springfield Hospital for the month of February is as follows:

Saturday, February 3.

9–10 a.m. Grand Surgical Rounds.
10–11 a.m. Radiology Conference in the Staff Conference Room. Topic to be announced. Dr. John W. Turner.
11 a.m.–12 m. Combined Medical-Surgical Grand Rounds in the Staff Conference Room.

Thursday, February 8.

12:15 p.m. Staff Meeting in the Staff Room.

Saturday, February 10.

9–10 a.m. Grand Surgical Rounds.
10–11 a.m. Seminar on Surgical Principles in the Staff Conference Room. "Vascular Surgery." Dr. F. C. Leonard.
11 a.m.–12 m. Clinicopathological Conference in the Staff Conference Room.

Wednesday, February 14.

12:15 p.m. Psychosomatic-Medicine Conference on the subject "The Psychoses, Organic and Functional" in the Staff Conference Room. Dr. Calvert Stein.

Saturday, February 17.

9–10 a.m. Grand Surgical Rounds.
10–11 a.m. Pathology Conference: Seminar on Hematology (continued) in the Staff Conference Room. Dr. William Kaufmann.
11 a.m.–12 m. Medical-Death Review in the Staff Conference Room.

Tuesday, February 20.

8:30 p.m. Guest Speaker Conference on the subject "The Surgery of Gastroduodenal Ulcers" in the Staff Conference Room. Dr. C. Stuart Welch, professor of surgery, Tufts College Medical School and surgeon-in-chief, New England Center Hospital, Boston.

Saturday, February 24.

9–10 a.m. Grand Surgical Rounds.
10–11 a.m. Seminar on Surgical Principles in the Staff Conference Room. "Rectal Surgery." Dr. Lewis Whiting.
11 a.m.–12 m. Surgical-Death Review in the Staff Conference Room.

SPRINGFIELD ACADEMY OF MEDICINE

A meeting of the New England Diabetic Association will be held at the Springfield Acadamy of Medicine on Tuesday, February 13, at 8:30 p.m. There will be a symposium on diabetes mellitus, followed by a panel discussion, with Dr. Alexander Marble as moderator.

PROGRAM

Dietary Regulation and Instruction. Dr. Frank N. Allan.
Methods of Detection and Diagnostic Studies. Dr. Harry Blotner.
Treatment of Diabetic Coma. Dr. Reed Harwood.
Ambulatory Treatment in Office and Out-Patient Department. Dr. David Hurwitz.
Surgery in Diabetes. Dr. Leland McKittrick.
Importance of Control in Prevention of Degenerative Complications. Dr. Howard Root.

NATIONAL GASTROENTEROLOGICAL ASSOCIATION 1951 AWARD CONTEST

The National Gastroenterological Association announces its annual cash prize award contest. One hundred dollars and a certificate of merit will be given for the best unpublished contribution on gastroenterology or allied subjects. Certificates will also be awarded those physicians whose contributions are deemed worthy. Contestants residing in the United States must be members of the American Medical Association. Those residing in foreign countries must be members of a similar organization in their own country. The award is to be made at the annual convention banquet of the National Gastroenterological Association in September, 1951. Certificates awarded to other physicians will be mailed to them. The decision of the judges will be final. The Association reserves the exclusive right of publishing the winning contribution, and those receiving certificates of merit, in its official publication, *The Review of Gastroenterology*. All entries for the 1951 prize should be limited to 5000 words, typewritten in English, prepared in manuscript form and submitted in five copies accompanied by an entry letter. Entries must be received not later than June 1, 1951. Entries should be addressed to the National Gastroenterological Association, 1819 Broadway, New York 23, N. Y.

AMERICAN PSYCHOSOMATIC SOCIETY

The eighth annual meeting of the American Psychosomatic Society will take place at Chalfonte–Haddon Hall, Atlantic City, New Jersey, on April 28. Both morning and afternoon sessions will feature papers on various topics.

The registration fee for nonmembers of the Society will be $2.00. The fee for students, interns, residents, fellows, and those in full-time academic positions will be $1.00; proper identification should be presented. There is no fee for members of the Society. Registration will begin at 8:00 a.m. on April 28. The meeting will be held in the Viking Room at Haddon Hall, and will begin at 9:00 a.m.

Further information may be secured from the office of the Society, at 714 Madison Avenue, New York 21, N. Y.

SOCIETY MEETINGS AND CONFERENCES

FEBRUARY 3. Massachusetts Society of Anesthesiologists. Page 160, issue of January 25.
FEBRUARY 3–24. Springfield Hospital. Notice above.
FEBRUARY 6. Boston City Hospital House Officers' Association. Page 160, issue of January 25.
FEBRUARY 8. Leukemia. Dr. Joseph Ross. Pentucket Association of Physicians. 8:30 p.m. Haverhill.
FEBRUARY 9. Beta Iota Chapter of Nu Sigma Nu. Page 1018, issue of December 21.
FEBRUARY 9. Cutter Lecture. Page 116, issue of January 18.
FEBRUARY 11. National Conference on Medical Services. Page 38, issue of January 4.
FEBRUARY 12–14. American College of Allergists. Page 74, issue of January 11.
FEBRUARY 13. New England Society of Anesthesiologists. Page 512, issue of September 28.
FEBRUARY 13. Harvard Medical Society. Page 197.
FEBRUARY 13. Springfield Academy of Medicine. Notice above.
FEBRUARY 14. Beta Kappa Chapter of Nu Sigma Nu. Page 197.
FEBRUARY 20. South End Medical Club. Notice above.
FEBRUARY 21. Bay State Society for the Crippled and Handicapped. Page 1066, issue of December 28.
FEBRUARY 27. Norfolk District Medical Society. Page 629, issue of October 19.
MARCH 5–8. New Orleans Graduate Medical Assembly. Page 1018, issue of December 21.
MARCH 7. Hampshire District Medical Society. Page 170, issue of July 27.
MARCH 11–16. College of Medical Evangelists Page 74, issue of January 11.
MARCH 22. Hyman Morrison Lecture. Page 74, issue of January 11.
MARCH 26. Boston Lying-in Hospital Obstetric Round-Table Conference. Page 629, issue of October 19.
MARCH 27. Norfolk District Medical Society. Page 629, issue of October 19.
APRIL 9–13. American College of Physicians. Page 296, issue of June 15.
APRIL 10. New England Society of Anesthesiologists. Page 512, issue of September 28.
APRIL 24. Norfolk District Medical Society. Page 629, issue of October 19.
APRIL 28. American Psychosomatic Society. Notice above.
MAY 8. New England Society of Anesthesiologists. Page 512, issue of September 28.
MAY 9. Norfolk District Medical Society. Page 629, issue of October 19.
MAY 16. New England Obstetrical and Gynecological Society. Hillcrest Country Club, Leicester, Massachusetts.
MAY 16–18. American Association of Genito-Urinary Surgeons. Page 160, issue of January 25.

(*Notices concluded on page xiii*)